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THE DEVELOPMENT OF THE AUDITORY OSSICLES AND ASSOCIATED STRUCTURES IN MAN

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In aquatic and amphibious vertebrates, the skeletal elements which in higher forms become transformed into ossicles function as branchial supports for the respiratory mechanism. These elements are made over to serve as links in an ossicular chain—converting air waves into liquid waves and these into auditory impulses.

Although it is in general true that most investigators are in agreement on a branchial derivation for the malleus and the incus, opinions differ widely as to the origin of the stapes. Since these matters have been recently discussed in some detail by Cauldwell and Anson,[2, 9] a brief review of opinions will suffice here.

Several early authorities maintained that the stapes originates from the first branchial arch, while others ascribed the total origin

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to the second visceral (hyoid) arch. This arch, or bar, was described as being attached originally to the chondrocranium in the region of the postsphenoid; with disappearance of this part, the remainder of the bar formed the stapes proximally and, in succession, the stapedial muscle, the pyramidal eminence, the styloid process, the stylohyoid ligament and the lesser cornu of the hyoid bone.

Opposed to the view which identifies the stapes with either the first or the second arch or bar is that which has it derived from the otic capsule itself. At least one author has contended not only that the stapes arises from the primordial skeleton of the embryonic head (and, therefore, from the same mesenchymal source that gave rise to the labyrinthine capsule) but also that the malleus and the incus were likewise derived from the capsular tissue. Others support the notion of a composite origin, entailing stapedial derivation partly from the second visceral bar and partly from the otic capsule.

Visceral and somatic skeletal elements take no part whatsoever in the development of the stapes, according to the opinion of some investigators. In their opinion the stapes arises independently of the other ossicles and of the otic capsule, having its inception in a circular mass of cells surrounding the stapedial artery. The stapes, then, while histologically similar to the adjacent incus and otic capsule, was considered to be of strictly independent origin, any contact with the hyoid bar or the otic capsule being an entirely secondary condition.

Many of these classical studies of ossicular derivation were carried out on comparative vertebrate material and involved the even more perplexing and basic problem of determining the primitive source of the branchial skeletal elements—of their homology with hyomandibular, quadrate and articular bones. In the current investigation attention has been paid only to developmental features in human embryos and fetuses—supplemented by such observations on postnatal stages as indicate adult retention of morphologic characters established in the ossicles long before the time of birth.

The present article is based upon a study of over 300 series of sections in the otological collections at the University of Wisconsin and at Northwestern University Medical School. From the combined lot of microscopic series, the following stages were selected for the illustrations: 28 mm. (8½ weeks); 111 mm. (15 weeks); 126 mm. (16¾ weeks); 135 mm. (17 weeks); 146 mm. (17½ weeks); 150 mm. (20 weeks); 180 mm. (23 weeks); 210 mm. (23 weeks); 222

mm. (25 weeks); 345 mm. (38 weeks).* In addition, the following later stages are represented in the figures: 18-year, 57-year and 70-year adults.

The reconstructions, prepared from Edinger tracings, were fabricated in cardboard (28 mm. at a magnification of 62½ diameters, 135 mm. and 161 mm. at 20 diameters); or in wax (150 mm., 180 mm., 210 mm., 18 years, 57 years and 70 years at 125 diameters). The line drawings of microscopic fields are based upon individual tracings prepared by use of the Edinger projection apparatus (at an original magnification of 50 diameters).

OBSERVATIONS AND DISCUSSION

As observed during an earlier phase of this general study of the human ear and temporal bone, the malleus and the incus are primordially related to the branchial, or visceral, arches, or bars; the stapes, on the contrary, has acquired—or, perhaps, primitively possessed—a relation which is as much capsular as it is branchial. These developmental features have already been described in journal articles† (esp., Cauldwell and Anson[2, 9]) and here require nothing more than brief review.

A. Early Development. The cytoblastema of the stapes appears in the 7-mm. embryo as a concentration of mesenchymal cells situated at the cranial end of the second branchial, or hyoid, bar (Reichert's cartilage). It is continuous with the hyoid bar, but separate from an inconspicuous condensation of mesenchyma which, in surrounding the otocyst, is the primordial tissue of the otic capsule.

At the 10.6-mm. stage expansion of both the stapedial and the capsular tissues has brought these two primordia into fairly close relationship, yet they remain distinguishable. The stapes is penetrated by a relatively large stapedial artery.

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*In the figures illustrating histological features the following series in the Wisconsin collection were employed: Fig. 2, Embryo 158, Left side (*a*, slide 7, section 15); *b*, sl. 7, sec. 1; *c*, sl. 6, sec. 9; *d*, sl. 6, sec. 2; *e*, sl. 5, sec. 11; *f*, sl. 2, sec. 15; Fig. 3, Embr. 50.L (*a*, sl. 28, sec. 1; *b*, sl. 24, sec. 1; *c*, sl. 18, sec. 4); Fig. 4; Embr. 11.L (*a*, sl. 34, sec. 1) and Embr. 5.L (*b*, sl. 33, sec. 2); Fig. 6, Embr. 30.L (*a*, sl. 25, sec. 8; *b*, sl. 21, sec. 10; *c*, sl. 14, sec. 10); Fig. 7, Embr. 46.L (*a*, sl. 60, sec. 1; *b*, sl. 53, sec. 3; *c*, sl. 50, sec. 1; *d*, sl. 31, sec. 1); Fig. 8, Embr. 61.L (*a*, sl. 71, sec. 1; *b*, sl. 44, sec. 1).

†Chiefly based upon an examination of series in the Carnegie Collection (Baltimore), which series need not be listed here. In the article referred to, photomicrographs of sections were used as illustrations. It is planned to prepare reconstructions of crucial stages; only by such means can continuities of fluctuating tissues and interrelationships of shifting structures be established to the conviction of a writer and be pictured to the satisfaction of a reader.

In the 13.1-mm. embryo, while the future capital part of the stapes is still continuous with the hyoid bar, Meckel's cartilage has just appeared.

At 13.95 mm. the stapes has attained definitely annular form; the heavier mesenchymal tissue along the internal margin of the obturator foramen is histologically distinct from that of looser texture through which the stapedial artery courses. The periotic and the stapedial masses are now in close apposition. The mesenchymal concentration which will become the first branchial arch (Meckel's cartilage) is now as clear as that which forms the stapes.

In the 16-mm. embryo the hyostapedial connection persists; from the combined tissue will later be derived the stapedius muscle and its tendon. Both the malleus and incus are now definable. The incus approaches, but not yet touches, the lateral or, future capital, extremity of the stapes; the medial or basal portion of the stapes is applied closely to the otic capsule, where the latter is concave for its reception. The existence of a concavity is owing chiefly to development of the angular mass, a portion of the capsule destined to house the semicircular ducts. At this stage a separation of the malleus and incus is initiated. The tissue of the ossicles and of the capsule is beginning to change into precartilage.

In the 17-mm. embryo further progress in the transition from mesenchyma to precartilage is generally evident. For the first time the incus and the stapes are in virtual approximation, being separated only by an ill-defined zone of mesenchymal cells. The malleus is distinguishable from Meckel's cartilage only through the presence of a slight constriction between the two—the malleate mass appearing as a bulbous enlargement (with caudally directed primordium of a manubrium) of the dorsal extremity of the branchial cartilage.* Ventrally the cartilages of Meckel have not met at the midline. Even at this early stage, when the branchial elements are still in the precartilaginous condition, the future mandible is represented, on each half of the embryonic head, by a leaf-like plate of true bone; these osseous plates lie externally to the ventral ends of the branchial arches, and are approximately one-third as long as the latter (compare 28 mm., Fig. 1). The incus seems merged with the medial portion of the malleus, the two masses being distinguishable through the presence, between them, of the chorda tympani. Reichert's cartilage is connected with the stapes by a band of precartilage. Of the three

*For this stage the features are based not only upon a study of sections but upon examination of a reconstruction (not herein figured) of the 17-mm. Bardeen embryo (Wis., Embr. 10).

ossicles the stapes is most independent and its form clearest; it is definitely ringshaped with a clearly defined obturator foramen.

In the 19.8-mm. stage this connection is maintained through a dense mesenchymal tissue, which is the forerunner of a hyostapedial ligament, from which will be derived the stapedius muscle and tendon. The malleus and incus are now made up of a young, true cartilage, while the stapes is still in a stage of transition between dense mesenchyma and precartilage.

In th 22-mm. and 23.4-mm. embryos the stapes has changed to precartilage. The hyostapedial tissue still forms a strong mesenchymal band connecting the lateral, or capital, portion of the stapes and the capsular extremity of the hyoid bar.

At the 25-mm. stage the stapes appears to be pressed into the lateral wall of the otic capsule, from the precartilaginous tissue of which the basal aspect of the stapes (consisting of similar tissue) is separated only by a thin seam of mesenchyma. For the most part, the capsule is precartilaginous, except where, in surrounding the epithelial labyrinth, its tissue is undergoing retrogressive change to allow for formation of the periotic spaces. The lateral aspect of the stapedial "ring" is slightly protruberant, and therefore distinguishable as the future capital portion; the medial aspect, now moderately flattened, is recognizable as a developing basal part. A rudimentary stapedial artery is still present. Both the malleus and incus are composed of young cartilage cells. The malleus is broadly continuous with the first branchial bar (Meckel's cartilage), but is histologically independent of the incus at the site of the developing malleo-incudal articulation. The area of the future incudostapedial joint is similarly identifiable. The site of the vestibular window is recognizable as the portion of the lateral capsular wall which is thinned and indented to receive the base of the stapes.

In embryos of 27.5 mm., 28 mm. and 30 mm. the otic capsule and the stapes are undergoing similar change to true cartilage, with the important exception of the lamina stapedialis against which the stapes rests. In the 28-mm embryo the malleus is still broadly continuous with Meckel's cartilage, in relation to which the bone of the mandible is developing rapidly (Fig. 1*a* and 1*b*). There is no cartilaginous connection between Reichert's cartilage and the incus.

In the 34-mm. embryo, the base of the stapes has undergone further flattening. Its external fibroblastic layer seemingly fuses with similar tissue of the lamina to become, conjoined, the new formative layer for the base of the stapes. The laminar portion is chiefly

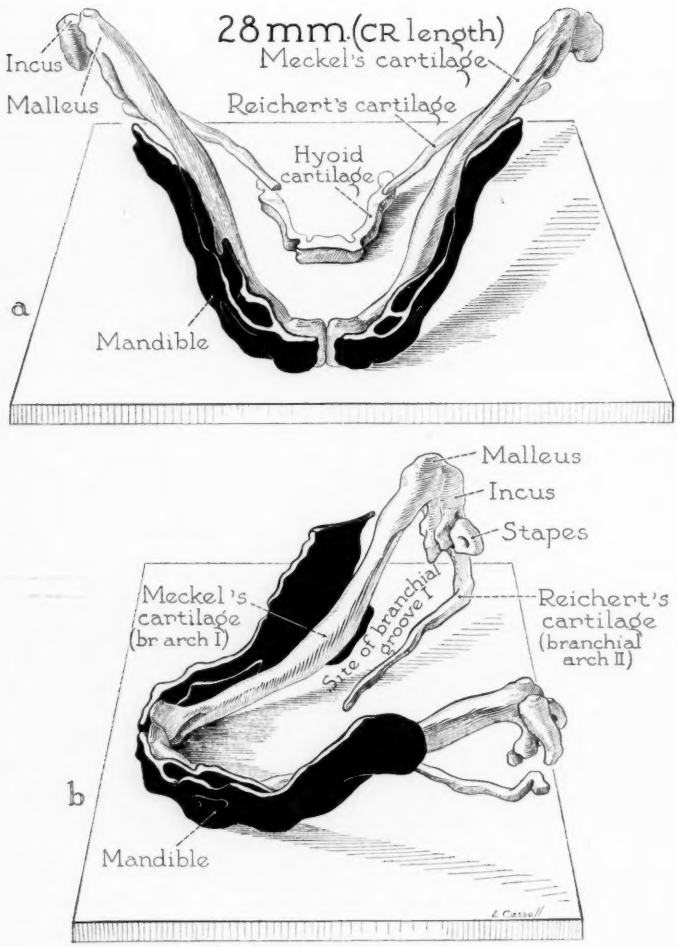

Fig. 1.—Reconstruction of cartilaginous ossicles and related branchial arches and osseous mandible in 28-mm. (CR length) embryo (Wisconsin No. 158). X 11 (approx.). *a*, anterior view; *b*, superolateral view (hyoid cartilage omitted).

instrumental in increasing the width and thickness of the vestibular aspect of the stapes and, thereby, in converting a convex into a flattened basal portion. And to that extent, too, it may be said that a localized area of the capsular wall contributes to the formation of the stapes. It is a point of interest that in some favorably stained series the original annular portion (deeply stained) of the stapes is set off sharply from the capsular contribution (lightly stained).

At 50 mm. the capital extremity of the stapes is foveate for reception of the incus; the crura are prominently bowed.* On the base, the transitional zone of earlier stages (between base and capsular lamina) has been obliterated, the deeper, or vestibular, lamella of the layer then becoming a secondary, true perichondrium of the base. Since this zone—the site of the future annular ligament—is merely one in which chondral matrix is present in smaller amount than in either the adjacent tissue of the base or that of the fenestra, it may properly be said that the stapes is histologically continuous with the vestibular "window," and that, in the sense in which the term fenestra is employed to describe the opening in the adult bone, no window exists in the otic capsule of the early fetus. Regarding the exact manner of formation of the annular ligament no conclusive statement can be made; because perichondrium common to the widening stapedial base and to the concurrently retreating fenestral rim is in a state of histological flux, it is difficult to determine whence the mesenchymal tissue of the developing ligament is recruited. But it can be said that, when the future site of the ligament becomes clearly defined, the tissue is mesenchymal and that the constituent cells are radially arranged.

In conclusion it may be said that according to the observations made during the present study the stapes in man is derived from two sources, namely, the second visceral (hyoid, Reichert's) bar and the lateral capsular wall, that the incus is primordially continuous with the same bar, and that the malleus is one with the first visceral bar (Meckel's cartilage).

The malleus and incus, as above noted, become areas of mesenchymal concentration somewhat later in embryonic development than does the stapes. However, in respect to the initiation of bone formation their development is precocious, since ossification begins in the incus and malleus at the 117-mm. and 126-mm. stages, respectively, whereas an ossification center does not appear in the stapes until the 146-mm. stage has been reached.

*This stage, two earlier stages (25 mm., 40 mm.) and nine of more advanced development are illustrated in Fig. 1 of an earlier article.[2]

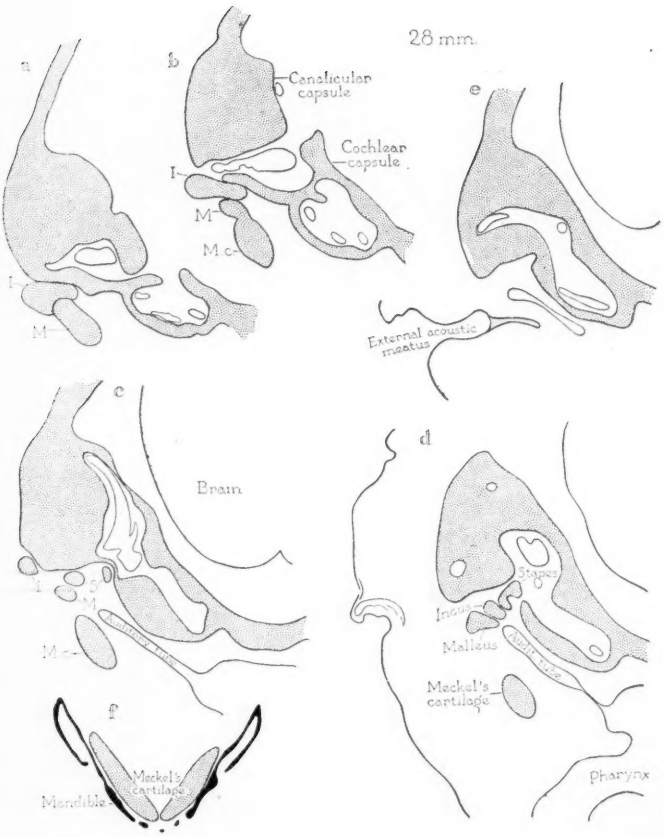

Fig. 2.—Tracings of sections showing ossicles and cartilaginous otic capsule in 28-mm. embryo (Wisconsin No. 158). X 11. I, incus; M, malleus; M.c., Meckel's cartilage; S, stapes.

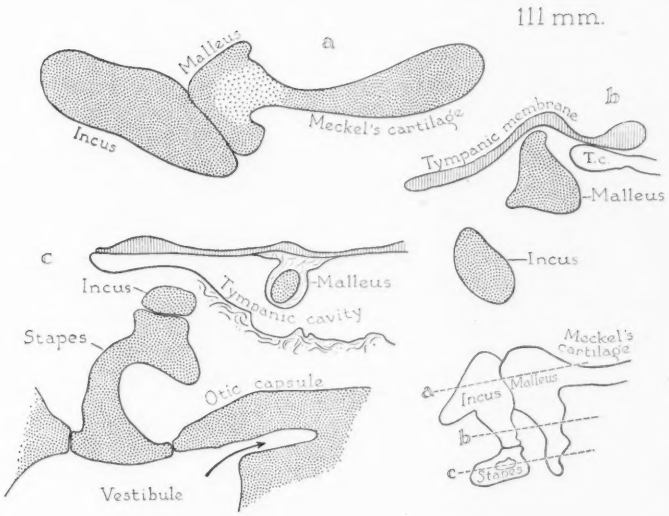

Fig. 3.—Tracings of sections continued; 111-mm. fetus (Wisconsin No. 50). X 13. *a*, *b* and *c* are sections at levels indicated in inset (respectively: articulation of malleus and incus; manubrium of malleus and long crus of incus; stapes). Mature cartilage is indicated by regular stippling, rarefied tissue in lighter texture. Arrow in *c* points into fissula ante fenestram (vestibular orifice).

B. Later Development. By way of review it may be pointed out that in the 28-mm. embryo the ossicles and associated Meckel's bar (first branchial arch) are fully formed in true cartilage (Fig. 1). The malleus is continuous with Meckel's cartilage, but the incus is separate from Reichert's cartilage (second branchial arch). The stapes is, at that stage, independent of all branchial elements. While no osseous tissue has yet appeared in any of the ossicles or in Meckel's bar, the latter is associated with, but not yet enveloped by, the developing bone of the mandible. At their ventral extremities the cartilages of Meckel are contiguous (Figs. 1*a* and 1*b*; Fig. 2*f*); where they meet they are somewhat flattened and each is elevated in the form of a crest (Fig. 1*b*); each is robust. The cartilages of Reichert, on the contrary, are slender (Fig. 1*a*); they do not meet ventrally and could be regarded as reaching the midline only through the intermediation of the hyoid cartilage, whose form begins to suggest that of its adult derivative. In comparison with the mandible, the

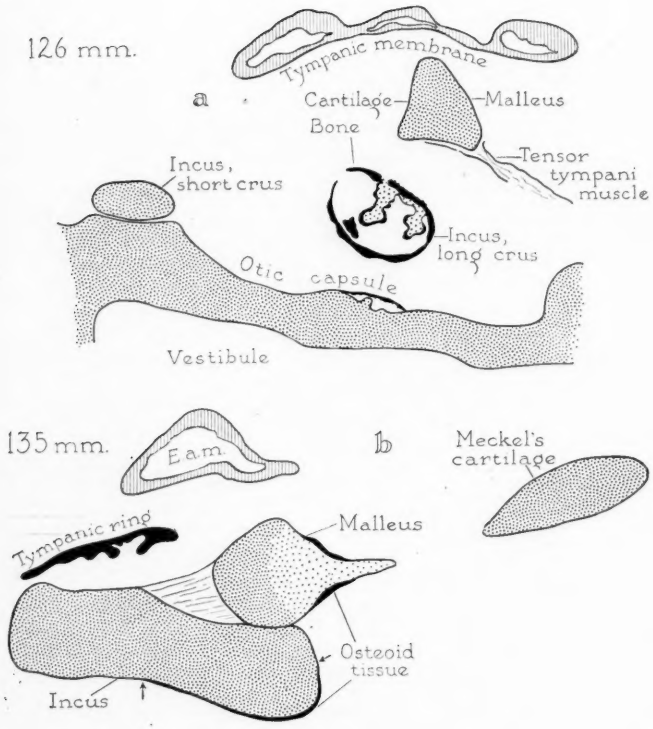

Fig. 4.—Tracings continued; *a*, 126-mm. fetus (Wisconsin No. 11); *b*, 135-mm. fetus (Wisconsin No. 5). X 15. Arrows point to margins of perichondral bone. E.a.m., external acoustic meatus.

branchial elements and their ossicular associates are laggards in development—the osseous mandible having not only spread along three-fourths of the length of Meckel's cartilage but having formed on its gingival edge sockets for the first teeth (Figs. 1*a* and 1*b*). At this stage the capsule is entirely cartilaginous (Figs. 2*a* to 2*f*).* The external acoustic meatus and the auditory tube are approaching the ossicles from auricular and pharyngeal areas (Figs. 2*c* and 2*e*).

In the 100-mm. fetus the ossicles are still cartilaginous. They remain so in the 111-mm. stage. However, in the malleus vacuolization of the cartilage is under way, at the point of continuity with Meckel's cartilage (Fig. 3*a*). Neither the cartilage of the manubrium of the malleus nor that of the long crus of the incus has undergone change (Figs. 3*b* and 3*c*). The capital end of the stapes is foveate for reception of the incus, the basal portion is flanged circumferentially for reception into the vestibular window, and the crus is bowed (Fig. 3*c*). All of these morphological features forecast the adult form of the stapes. The otic capsule is entirely chondral. In it appears prominently the fissula ante fenestram (Fig. 3*c*, arrow into vestibular orifice). The pharyngeal mucous membrane has pressed forward to the point where it covers the tympanic membrane, is draped over the manubrium of the malleus and is advancing medially toward the stapes and otic capsule (Fig. 3*c*).

First ossification occurs in the incus; a thin shell of bone appears, in the 117-mm. fetus, on the anterior aspect of the long crus. At the 126-mm. stage bone has spread to form a shallow collar around the long crus (Fig. 4*a*), and to extend superiorly to the anterior surface of the body of the ossicle. In the same specimen bone makes its initial appearance on the malleus, but merely as a pellicle on the medial surface of the body near the area of articulation with the incus. In the 135-mm. fetus bone is present on the lateral as well as the medial surface of the malleus where the latter is continuous with Meckel's bar (Fig. 4*b*). Of these two earliest sites of ossification, the incudal is the more advanced: within the long crus cartilage has been almost totally removed inside of the shell of perichondral bone (Fig. 4*a*); within the body of the malleus cartilage is merely rarefied and undergoing calcification beneath the thin layer of osteoid tissue (Fig. 4*b*). Ossification has been initiated in the otic capsule at the

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*The otic capsule of a closely similar stage (29-mm. embryo, Harvard Collection) has been described and figured (with the use of reconstructions) by Martin and Anson.[5] See also Macklin.[10, 11]

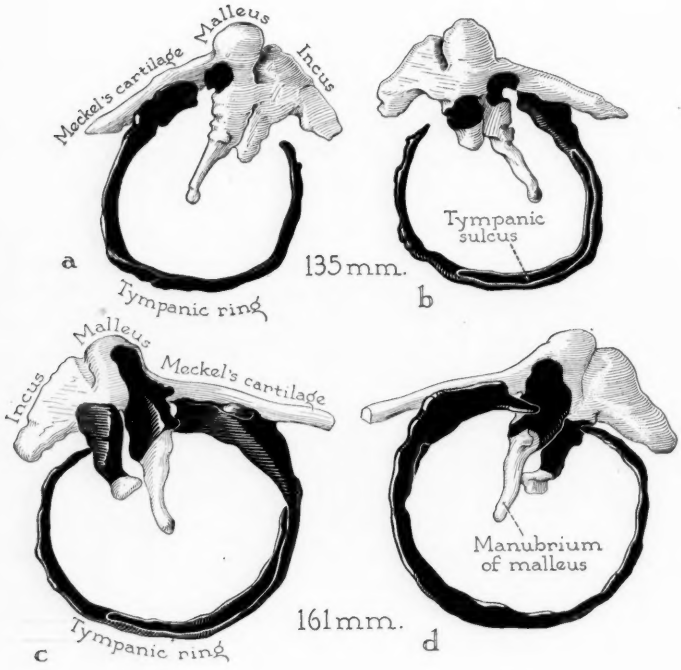

Fig. 5.—Reconstructions of malleus (and Meckel's cartilage), incus and tympanic ring in fetuses of 135 mm. (Wisconsin No. 5) and 161 mm. (Wisconsin No. 13). X 9. Left side. *a* and *d*, lateral aspect; *b* and *c*, medial aspect.

126-mm. stage (Fig. 4*a*);* the tympanic ring is well formed in bone (Fig. 4*b*). In Meckel's cartilage pre-osseous changes are evident only in the area where the latter is continuous with the malleus (Fig. 4*b*).

As seen in the 135-mm. fetus the centers of ossification in the incus and malleus are localized plaques (Figs. 5*a* and 5*b*); neither encircles its ossicle. Concurrently, the tympanic ring is an almost complete annulet of bone; it is guttered for attachment of the tympanic membrane (sulcus at end of leader in Fig. 5*b*).

*For details of ossification in the otic capsule the reader is referred to the article by Bast,[7] which figures reconstructions prepared from series utilized in the current study.

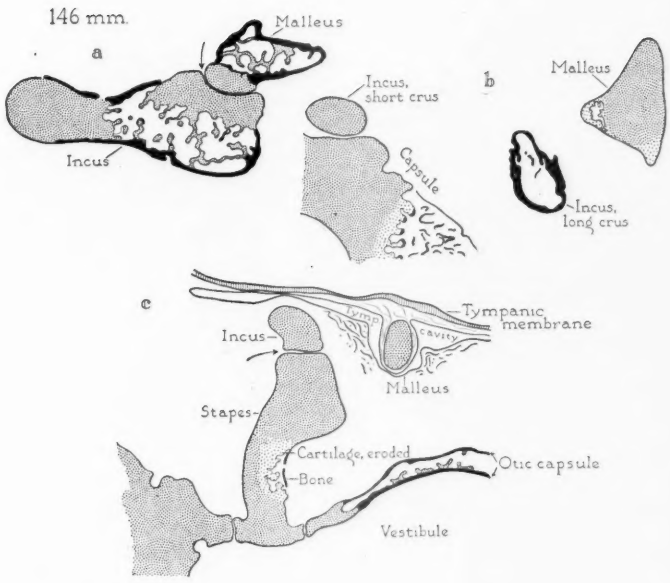

Fig. 6.—Tracings continued (at levels corresponding to those in Fig. 3), in 146-mm. fetus (Wisconsin No. 30). X 11. Arrows point to articulations.

Not until the fetus has reached the 146-mm. stage does ossification of the stapes begin. The single center appears on the obturator surface of the base and spreads therefrom along each crus (Fig. 6*c*, posterior crus) toward the head of the ossicle. In the body of the incus, bone has largely replaced cartilage (Fig. 6*a*); the long crus is an osseous shaft, but the short crus is still cartilaginous (Fig. 6*b*). The head of the malleus now resembles a miniature long bone, being composed of a periosteal wall enclosing a marrow cavity (Fig. 6*a*); however, the manubrium is still largely made up of unaltered cartilage (Fig. 6*c*). Considering, then, the sites of primary ossification in the three ossicles, it may be said that, at the 146-mm. stage, while the merest pellicle of bone is present on the stapes (obturator surfaces of the base and of the adjacent areas of the crura), an appreciable layer of bone is found on the malleus (body, where the malleus joins Meckel's cartilage) and a complete shell of perichondral bone, with absence of original cartilage, forms the long crus of the incus. The

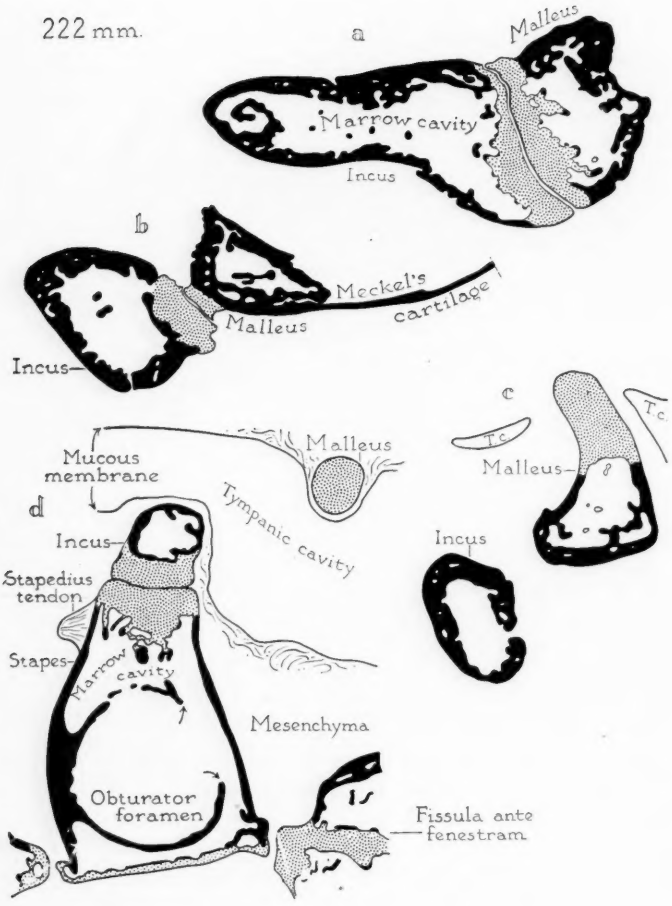

Fig. 7.—Tracings continued; ossicles, etc., in 222-mm. fetus (Wisconsin No. 46). X 14. Meckel's cartilage is partially shown; unlabelled arrows in *d* indicate edges of eroded obturator wall.

ossicles might be expected to hold relative positions in developmental progress until fully formed in bone. Actually, the incus does so in relation to the malleus; the stapes, as will be described, follows a separate path in its morphogenesis. Development of bone in the otic capsule anterior to the vestibular window (Fig. *6c*) has reached a stage comparable to that obtaining in the bodies of the malleus and incus (Fig. *6a*).

Bone spreads from each of the solitary ossification centers; in case of the incus, in the 161-mm. fetus, all but the tip of the long crus of the incus is formed in bone, while in the case of the malleus bone has spread downward on the manubrium and upward on the body of the ossicle (Figs. *5a* and *5b*).

In the 183-mm. fetus the crura of the stapes have become osseous columns hollowed to contain tissue; bone has spread to the tympanic aspect of the base, but the vestibular side is, and will remain, cartilaginous; the head of the stapes is a cylinder of solid cartilage. The malleus is largely ossified, being composed of a thin shell of periosteal bone enclosing a marrow cavity. The long crus of the incus has advanced to a similar stage of development. Meckel's cartilage is still continuous with the malleus.

In the 222-mm. fetus the body of the incus and the head of the malleus are osseous except in their articular portions (Figs. *7a* and *7b*). The long crus of the incus and the proximal portion of the manubrium of the malleus are also composed of bone (Fig. *7c*), but the distal extremity of the manubrium is cartilaginous (Fig. *7d*). Meckel's bar is now a long, saber-shaped rod of bone (Fig. *7b*). In the stapes excavation of bone on the obturator surface is under way to convert the crura into channelled and eroded members, the head into a hollow cylinder open to the obturator space and the base into a thinned bilaminar plate (Fig. *7d*). Cartilage still covers, and will persist on, the incudal aspect of the head and the vestibular surface of the base. Production of endochondral bone in the malleus and incus results in appreciable thickening of these ossicles, with concurrent reduction of their marrow cavities (Fig. *7a*). The stapes, on the contrary, has undergone thinning and striking reduction in bulk and will presently lose all of its marrow tissue. When this stage has been reached, the ossicles may be said to have set out on divergent developmental paths: the malleus and incus, like long bones generally, become thicker, their parts suggesting the diaphyses of regular bones; the stapes, on the contrary has begun to sacrifice its osseous and marrow substance and to prepare for invasion by mucosal tissue, which will ultimately come to rest against endochondral surfaces.

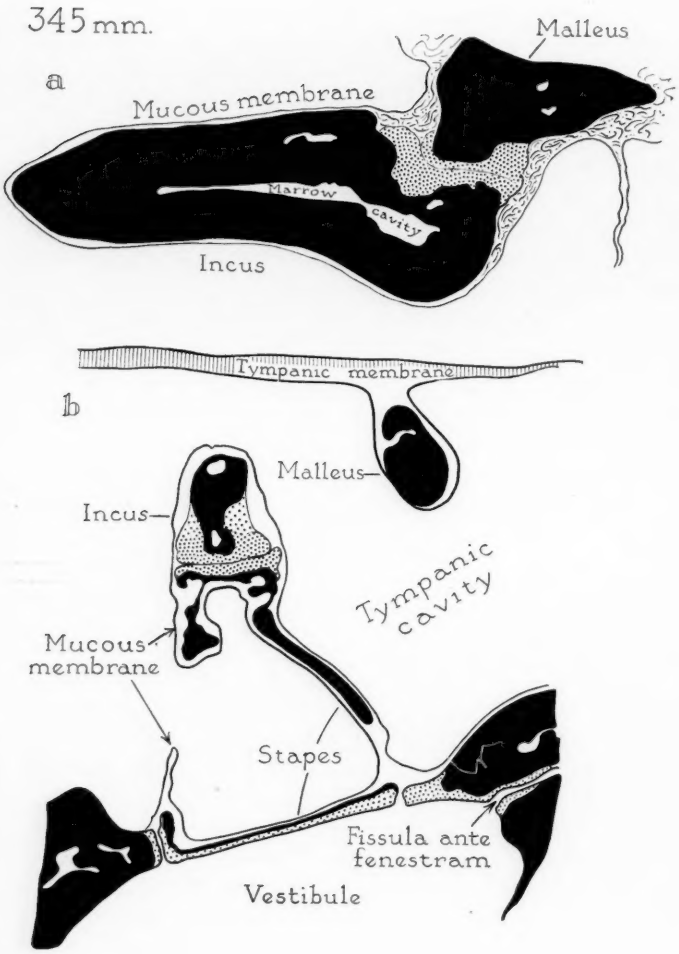

Fig. 8.—Tracings concluded; ossicles, etc., in 345-mm. fetus (Wisconsin No. 61). X 18.

It should be added that despite the differences just noted the ossicles are all alike in the matter of longitudinal growth; since they lack secondary, or epiphyseal, centers, they do not lengthen when once fully formed in perichondral bone. Cartilage will remain throughout life on the articular surfaces. In the 222-mm. fetus, at the incudomallear joint it still makes up a pair of thick chondral plates, with bone forming an osseous lamella on the internal aspect of each (Figs. 7*a* and 7*b*). At the incudostapedial joint bone is acting similarly (Fig. 7*d*); however, the new bone which, at this stage, is being produced at the capital extremity of the stapes will presently be resorbed, to be replaced by a very thin lamella situated closer to the articular surface of the joint (cf. Fig. 8*a*). The osseous plate will match, in disposition and fineness, the layer which has already spread across the base in the 222-mm. fetus (Fig. 7*d*). In the otic capsule, cartilage remains—and will persist throughout life—as a chondral rim lining the vestibular window. In this specimen, as in numerous other fetuses and infants, the cartilage of the fenestra is continuous with similar tissue which surrounds the fissula (Fig. 7*d*).

In the 345-mm. fetus the stapes has assumed adult form, as have also the malleus and incus (Figs. 8*a* and 8*b*). The stapes is deeply excavated on its obturator surface, mucous membrane being now draped over the channelled crura and eroded head and base, submucosal tissue replacing the original marrow (Fig. 8*b*). The malleus and incus, on the contrary, are made up almost solidly of bone; marrow is minimal in amount and is situated at the core of the bone (Fig. 8*a*); mucous membrane covers their external surfaces (Fig. 8*a*), does not invade, or replace, their cancellous tissue. In comparison with the other two ossicles, the stapes seems a veritable wraith; not only are its crura a mere twelfth the thickness of a process of the incus, but the stapedial head, or the base, is far thinner than the articular portion of either the malleus or the incus (cf. Fig. 8*b* with Fig. 8*a*). Owing to the production of intrachondral bone, the otic capsule assumes a "petrous" character (Fig. 8*b*). The cartilage of the fissular tract, almost completely replaced by bone in the 210-mm. fetus, remains for a time as perichondrium. Then as a result of renewed activity, the latter tissue produces new cartilage; this cartilage, representing a second phase of growth, lines the fissular tract (Fig. 8*b*). Thereafter it normally persists throughout an individual's lifetime at the vestibular extremity of the fissula ante fenestram, but is usually displaced at the tympanic end of the fissular tract.

The ossicles of the 367-mm (term) fetus are very similar to those of the 345-mm. stage. In the late fetal stage, then, adult form and

structure are attained by the ossicles and adult relationships established; the ossicular muscles, tensor and stapedius, are fully developed, and their tendons are implanted into the manubrium of the malleus and the neck of the stapes, respectively.

The crucial stages in morphogenesis of the stapes can be best summarized by following the developmental steps through which the stapedial crus, head and base pass to attain adult structure and form.*

Of the three major portions of the stapes, the crus follows the simplest succession. Each crus is, in the 100-mm fetus, a cartilaginous column. The hyaline cartilage is then altered and eroded by invasive buds of osteogenetic tissue; as a consequence, it is dissolved away while undergoing calcification (160-mm. fetus). Externally the formation of an osseous shell keeps pace with the progress of internal dissolution, so that a solid cartilaginous cylinder is converted into a hollowed bony tube whose cavity contains a primitive marrow but almost no endochondral bone (183 mm.). Up to and including this stage in morphogenesis, the regular processes of intracartilaginous ossification are at work in a familiar way; thereafter certain of these processes progress in an unusual manner and to unexpected degree. Thus, the periosteal shell of each crus, produced as in any long bone, undergoes extensive erosion on its inner, or obturator, surface with the result that one aspect of each of the crural tubes is rendered extensively foraminous. Bone of endochondral type is, at its maximum, so slight in amount and so transitory that the crura are almost completely periosteal in derivation, and their cavities devoid of cancellous bone. The periosteum ceases to be active as soon as the osseous shell is formed, so that, coupled with the want of accrescence from within, the crural tube does not increase in circumference. At neither end does an epiphyseal plate occur; increase in length is, therefore, also impossible. Terminally, each crus resembles a minature tibia halved longitudinally (term fetus). After the marrow cavity has been laid open by these erosive changes, mucous membrane, with submucosal tissue, replaces the embryonal marrow—in the way in which pharyngeal epithelium replaces diploic tissue in the formation of a paranasal air sinus.

The head of the stapes is likewise eroded from the obturator surface; however, removal of cartilage is not as rapid as it is in the case of the crus. Since the capital part of the adult stapes is a cylinder closed at its lateral, or articular end, fetal excavation must take place

*These steps are illustrated, employing segments of reconstructions, in Fig. 7, of an article by Anson, Cauldwell and Reimann.[4]

on the opposite, or obturator extremity. Cartilage (100 mm.) is eroded (183 mm.), and endochondral bone is formed in relative abundance through the conversion of calcified cartilage (180 mm., 245 mm.). The tips of the chondral hummocks within the core of the head are the first portions to be ossified. These islets of bone broaden and, concurrently with destruction of the cartilage upon which they rest, fuse to form a lamella of endochondral bone on the internal surface of the articular portion of the cartilage. Cartilage thus persists only on the articular surface, where it is lined by the thin layer of endochondral bone, to render that articular plate bilaminar (adult). Most of the irregular, excavated tissue is removed by this process to make a hollow head whose walls are usually smoothed. Here, as in the crura, marrow is replaced as the mucous membrane of the tympanic cavity invades the stapedial head. No epiphysis occurs; therefore, the head does not lengthen after it has been formed in bone. The periosteum of the head, like that of the crura, remains virtually inactive; consequently, the head does not widen. Therefore, the head of the stapes differs from the extremity of a typical long bone in lacking epiphysis as well as cancellous tissue and marrow, and in being covered on the interior by mucous membrane.

Developmental steps at the basal extremity make up an even more complex series. The ossification center, which spreads from the base along the crura to the head, first appears on the obturator surface of the base (150 mm.; cf. 100 mm.). On this lateral, or tympanic, surface profound structural changes occur, while the opposite, or vestibular, surface, together with the adjacent circumferential area for fenestral articulation, remains an unaltered oval plate of cartilage. As in the case of the head, the cartilage of the obturator aspect of the base is eroded, calcified, and converted into endochondral bone (160 mm.); then this newly-formed bone is largely removed. Duplicating the steps in the alteration of the head, the periosteal shell on the tympanic aspect of the base becomes extensively foraminous (183 mm.), and next is largely resorbed. When it has been rendered extensively perforate, the cupola-like "roof" of the base rather quickly becomes depressed (180 mm.), so that it is brought close to the "floor" (vestibular lamina). Over the floor of this marrow cavity endochondral bone spreads to produce first an irregular, and subsequently a fairly smooth, layer (183 mm. and 180 mm., respectively). The process of osteogenesis is complicated at this juncture by an act of developmental salvage, through the operation of which some bone of the roof is not removed, but, escaping erosion, remains to produce a peripheral rim for the base. In some specimens it also persists as an intercrural crest on the obturator

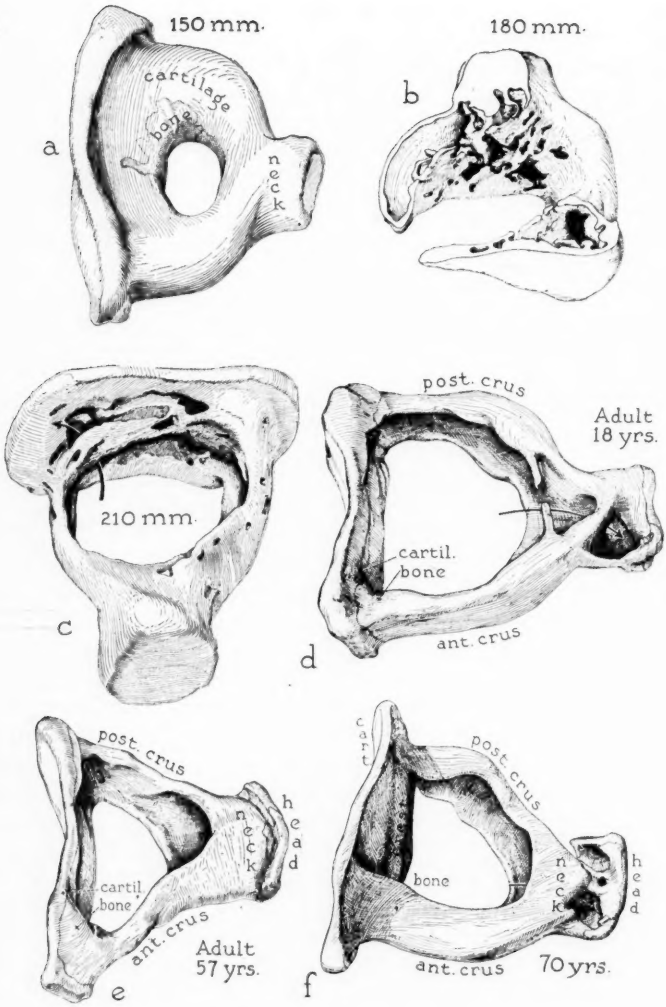

Fig. 9.—Reconstructions of stapes: *a*, 150-mm. fetus (Wisconsin No. 39); *b*, 180 mm. (Wisconsin No. 45B) with cranial portion of model removed to exhibit extent of excavation; *c*, 210 mm. (Wisconsin No. 51); *d*, adult of 18 years (Northwestern Ser. 2.26.30); *e*, adult of 57 years (Northwestern Ser. 1.14.33); *f*, adult of 70 years (Northwestern Ser. 3.3.34). X 18. Arrow in *c* passes beneath crista; arrow in *d* passes through excavated portion of neck and head of ossicle.

surface. The crest may remain as a low ridge in the late fetus (210 mm., term, at crosses) and in the adult. The relatively tall peripheral ledge—which, unlike the crest, is a constant feature—is composed of periosteal bone of the tympanic wall of the originally hollowed base, combined with endochondral tissue of the inner lamella of the two-layered vestibular wall. When these strata meet and fuse, some spaces of haversian character remain unobliterated and persist to serve for transmission of blood vessels from the newly-formed submucosa into the substance of the base (210 mm.; term). The intercrural crista is similarly constituted. Mucous membrane, pressing medialward to invest the outer wall of the otic capsule, covers the tympanic aspect of the stapedial base just as it came to invade the corresponding surfaces of the crura and the head.

The extent to which these morphogenic changes depart from the familiar scheme of bone development becomes more apparent when the formation of the stapes is compared with that of a long bone. In such a bone, the external configuration of the primordial cartilage model suggests in a general way the form of the same skeletal element as it is found in the adult body.* Bone formation begins in the diaphysis and surrounds the shaft in the form of an elongate collar. The perichondrium, upon conversion into periosteum, adds both length and thickness to the enveloping bony shell; concurrently, endochondral bone comes to replace the cartilage still remaining within the shaft. Bone formation is augmented by an auxiliary histological growth, namely, the secondary ossification center of each epiphysis. Spongious endochondral bone of the diaphysis is largely dissolved to produce the secondary, or permanent, marrow cavity. When the cartilage in the plates at the two extremities is finally replaced by bone, longitudinal growth ceases; in the tibia this change occurs in the distal epiphysis at the age of 17 and in the proximal one between the nineteenth and twenty-third year. Cartilage finally remains only on the articular surfaces of the two extremities.

In the stapes, on the contrary, at neither capital nor basal end does there occur a zonal mechanism for epiphyseal growth, and throughout the stapes periosteal deposition ceases as soon as osseous tissue, spreading from the lone center on the lateral aspect of the base (Fig. 9*a*), completely encircles the obturator space (Fig. 9*b*); cessation of growth takes place when the fetus has reached the halfway mark in its intra-uterine existence. The tibia grows without interruption until the individual is an adult; the stapes, on the contrary, is as large at mid-term (180-mm. stage) as it will ever be.

*Plate IV in Anson and Cauldwell.[2]

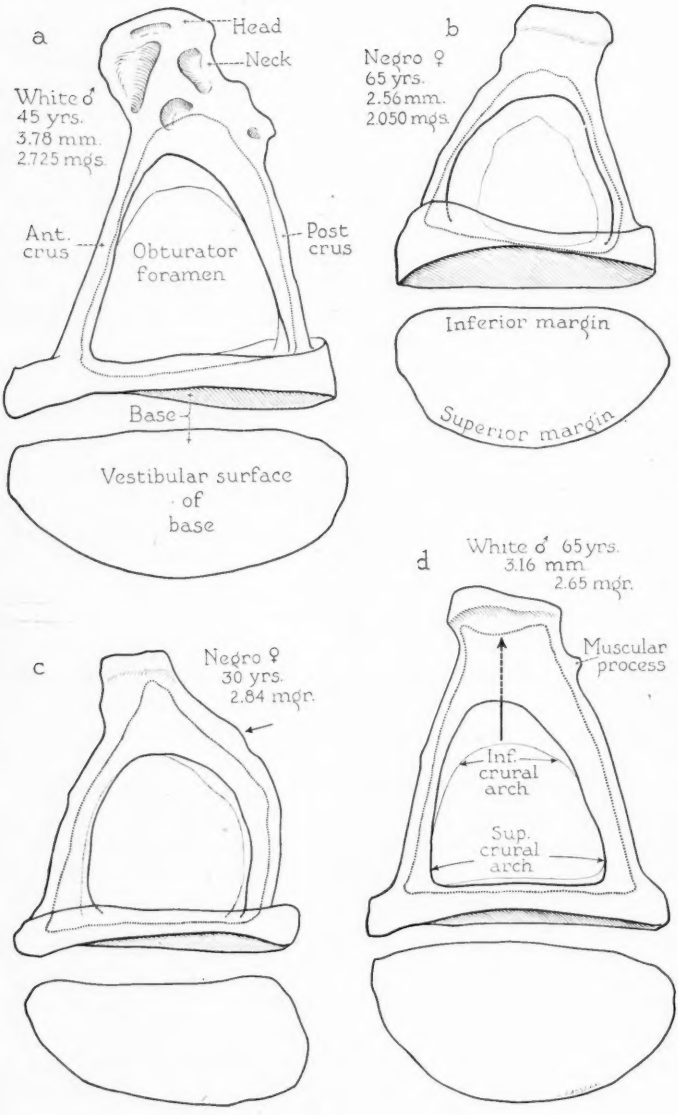

Fig. 10.—Drawings of excised stapes, selected to illustrate variation in form and size. X 15.

Attainment of full size by the tibia is a developmental achievement of early manhood; the stapes has accomplished as much before the fetus has reached the 5-month stage. And while the tibia of the adult is 36 times the length of the tibia in the 30-mm. embryo, the length of the stapes in the adult is only six times as great as that of the ossicle of the 25-mm. or 30-mm. stage.

At the 180-mm. stage the crura and base are hollowed (Fig. 9*b*). Concurrently with continuing ossification, the newly-formed bone is removed around the obturator aspect. The entire obturator wall in the 210-mm. fetus has been destroyed by osteoclasis (Fig. 9*c*). In the base, some of the perichondral bone persists as a marginal lip and in some specimens, as the transverse stapedial crest (Fig. 9*c*). At the 245-mm. stage, adult configuration is almost attained, is actually reached in the 275-mm. fetus. In the term fetus, infant, child or adult the head of the stapes may be perforated (Figs. 9*d* and 9*f*) or uneroded (Fig. 9*e*); the margin of the base may be lipped (Fig. 9*d*) or flattish (Fig. 9*f*), the crura bowed (Fig. 9*d*) or relatively straight (Fig. 9*e*).*

Thus, while in long bones ossification is initiated at or near the middle of the shaft to form a collar of primitive bone, in the stapes it is begun in the flattened basal part of what was primordially a ring of cartilage; therefrom it spreads along the crura and the neck, to become continuous around the obturator foramen. Since secondary centers of ossification are wanting, stapedial development resembles that obtaining within the diaphyseal portion of a long bone split lengthwise. As soon as these "diaphyseal" features are established, the ensuing formative stages become so specialized that they serve to mark the stapes as a unique skeletal element. With the formation of the periosteal shell, the mechanism is set in operation for completing the conversion of a solid chondral member, of modified annular shape, into a hollowed structure in which epiphyseal interruption is wanting. Thus it is, that while an adult tibia retains a complete external shell of compact bone with a core of cancellous osseous material and fatty marrow, a stapes loses approximately one-half its periosteal bone and virtually all of its endochondral tissue and associated marrow.

C. Adult Form. Variations are to be expected in the anatomy of the stapes of older persons, since, as already pointed out, they occur in the ossicles from advanced fetus, newborn and infant. In these,

*Reconstructions of stapes of these and of other stages are described and figured by Anson, Karabin and Martin[5, 6] and by Anson and Cauldwell.[3]

the neck of the stapes may be eroded or may remain a smooth-walled tubular segment; the tubercle for the stapedius tendon may be prominent or wanting, the medial ends of the crura narrow or broad at their points of continuity with the base and the length of the crura or the width of the base less in the stapes of an infant than in that of late fetus. All of these differences are within normal range of anatomic variation, being regularly encountered in ossicles (75 in current series) from adults between the ages of 25 and 75 years.*

The base of the stapes varies widely in outline (Figs. 10*a* to 10*d*). Although usually described as oval, elliptical or bean-shaped, such forms are not common. The usual shape is one in which the superior margin is generally convex, the inferior edge almost straight, the anterior and posterior borders rounded, with the anterior tending to be a little more pointed than the posterior. Uncommonly the inferior edge is concave, to produce a reniform base (Fig. 10*c*). On the other hand, this margin may be convex and, consequently, the whole outline oval. Frequently the angle between the anterior and superior margins is sharp, owing to straightness in the anterior part of the superior margin (Fig. 10*d*). Size variations make for all graduations between a short, wide base and a long narrow one (Fig. 10*a*). Generally the anterior end is either slightly larger than the posterior, or the two ends are equal. Occasionally the anterior end is pointed (Fig. 10*c*). The vestibular surface of the base is sometimes slightly convex; this has been regarded as a regular feature. Rarely the base is flat.

The tympanic surface of the base is, in most instances, concave; concavity is in part due to the presence of a peripheral ridge which is often prominent even in early fetal stages. This marginal elevation is complete in some specimens, and therefore merged with the edges of each crus (Fig. 10*d*). Since, in young specimens, continuity of the basal lip and the crural edge is always encountered, its occurrence in adult specimens may be regarded as retention of a fetal character. The crura almost invariably originate from points nearer the inferior than the superior margin of the base. On the tympanic surface of the footplate is found an inconstant ridge, the crista stapedis, which passes between the crura. It is present in less than one third of specimens, and even then is likely to be incomplete. It supposedly represents the line of attachment of the obturator membrane. This crest, as previously described, is a remnant of a once complete wall on the

*Other examples selected from this lot of excised specimens are figured by Beaton and Anson.[8] The plate reproduced in the current article is used with the permission of the Quarterly Bulletin of Northwestern University Medical School.

obturator surface of the base (cf. Fig. 9*c*). The average circumference of the base, in 75 specimens, is 7.45 mm, with extremes of 6.48 and 8.30; the average length is 2.99 mm. (extremes, 2.64 and 3.36); the average width is 1.41 mm. (extremes, 1.08 and 1.66).

The crura, arising from the extremities of the base, join laterally to complete the crural arch and to bound the obturator foramen. The shape of the arch is usually ovoid (Figs. 10*a* and 10*d*); rarely it is triangular with a sharp apex; quite often it is almost circular (Fig. 10*c*). Each crus is channelled in the adult, as it is in fetal specimens; the hollowing is on the inner, or obturator, surface (Figs. 10*a* to 10*d*, and 9*c* to 9*f*). By virtue of this peculiar structural feature, each crus has a superior and an inferior edge. The two superior edges meet at the capital end, as do the two inferior edges, so that, in reality, two arches are formed. The hollowed portion bounded by the edges then continues into the neck and into the head. In specimens with tall basal lips the base itself lends the appearance of being similarly excavated. The meeting of the crura to form the stapedial arch gives the stapes its distinctive shape, and depending on crural length and curvature, the shape of the obturator foramen is altered. The crura vary widely in size, from slender stems (Fig. 10*a*) to sturdy structures. Almost without exception the anterior crus is the slenderer; rarely is it the bulkier of the two and usually it is the straighter (hence the term, crus rectilineum). The posterior crus is curved (crus curvilineum). Exceptional specimens do occur in which the posterior crus is straighter than the anterior. Because of the difference in degree of curvature, the average length of the anterior crus (head to base) is 3.62 mm., that of the posterior crus, 3.73 mm. However, rare specimens are encountered in which the anterior crus is the longer of the pair. Frequently specimens occur in which the crura are approximately equal in length and curvature. The crura are subject to the formation of exostoses, as is any other part of the ossicle. When posterior crural in position, an exostosis may be mistaken for the muscular spine for the attachment of the stapedius muscle. The posterior crus occasionally exhibits a knee-like thickening at the junction of its lateral and middle thirds.

The obturator foramen owes its variation in shape chiefly to variation in the length, curvature and position of the two crura. Thus the foramen, and hence the stapes, may be long (Fig. 10*a*) or short (Fig. 10*b*). If the crura are of equal length and of similar curvature, the foramen and the stapes itself have the general form of an isosceles triangle. The usual shape (dependent on common differences in curvature) departs from the isosceles form: if the crura meet at an

acute angle, a pointed arch and foramen result, while if they meet obtusely, the arch is rounded (Fig. 10*c*). As has been observed by many authors, the superior crural arch is generally somewhat greater in circumference than the inferior (Fig. 10*d*). Rarely this size relation is reversed. The difference that usually exists between circumferences of the superior and inferior crural arches is due to the extent to which each was eroded in process of conversion from hollow cylinders to channelled members. The anterior arch is generally higher than the posterior and is likely to show a more acute angle than the posterior (Fig. 10*d*). At times the crural edges are almost parallel (Fig. 10*c*). These margins may end without spreading at the base, may expand to join the peripheral rim, or may be continued into the crista stapedis. Rarely the two superior or the two inferior edges are so tall at the basal extremity as to continue across the corresponding margins of the base in the manner in which they do in the fetal or infantile stapes (cf. Fig. 9*c*). The space of the obturator foramen then appears to be invading the tympanic surface of the base deeply, suggesting the regular extension of the same space into the capital part of the ossicle. When seen in the natural condition, these osseous hollowings are, of course, masked by mucous membrane.

A stapedial neck is usually definable (Figs. 10*a* and 10*d*); exceptionally, a neck cannot be identified, the crura blending insensibly with the head (Fig. 10*c*). The neck is subjected to early bone resorption, a process which, seemingly, may continue slowly throughout life. The external configuration of the neck is strikingly variable. Extensive external pitting produces foramina, bony ridges, or marked thinning (Figs. 9*d* and 10*a*). Resorption may, on the contrary, be minimal, the neck then remaining as a smooth osseous structure (Figs. 9*e* and 10*d*). Since the hollowing of the crura usually extends into the capital subdivision of the stapes, the neck in some specimens is a hollow shell. Rarely the neck is but slightly excavated internally, remaining a solid cylinder (Fig. 10*b*).

The stapedius muscle is attached to the neck of the ossicle in over 75 per cent of cases; in other instances it is inserted into the head or into the "shoulder" of the posterior crus (Fig. 10*c*, at arrow). The area of attachment may be smooth, merely a roughened area (Fig. 10*c*), or a spine of fair prominence (Figs. 10*a* and 10*d*). The spine may be situated on the neck (Fig. 10*a*) or on the head. A depression at the point of attachment has been described by some authorities.

The head is perhaps the most variable portion of the ossicle. Variation is owing to difference in degree of bone resorption, to the

presence or absence of the processus muscularis, and to differences in the depth and outline of the fovea for reception of the lenticular process of the incus. The head may be very small (Fig. 10*c*), large (Fig. 10*d*), or of intermediate size. External erosion may extend into the head (Fig. 10*a*), as may also internal hollowing (Fig. 10*d*, to point of arrow). The presence of a processus muscularis affects conformation of the margin of the posterior surface. The depression for reception of the lenticular process may be foveate, irregularly grooved, triangular, or quadrilateral. The head of the stapes usually meets the crura at a slight angle, being tilted superiorly. Inferior tilting is uncommon. In many cases the planes of head and crura are in a straight line. The head is usually turned anteriorly to a slight degree (Fig. 10*a*), rarely posteriorly; sometimes the head meets the crural junction without curve (Fig. 10*d*).

Great variation exists in stapedial size (compare heights, Figs. 10*a* and 10*b*). The average height of the stapes is 3.26 mm., with extremes of 2.50 and 3.78 mm. There is also great variation in weight; the average weight is 2.860 mg., with extremes of 2.050 and 4.350 mg.

CONCLUSIONS

Omitting repetitious summary of the more detailed observations hereinbefore presented, the following general statements may be made by way of conclusion:

1. The ossicles in man follow a remarkable series of developmental steps in attaining adult form—a circumstance which might be expected when it is considered that they are made over to serve, in higher vertebrates, a function very different from their primitive office.

2. The malleus and incus are developmentally less aberrant than the stapes. Yet they differ from bones generally, not only in possessing peculiar form, but also in lacking epiphyseal centers of growth, in having a single ossification center each, and in attaining adult size and shape in the fetal body.

3. The stapes, in addition to displaying the features named above, is unique in the following respects: each crus (corresponding to the shaft of a long bone) becomes channelled to resemble a long bone halved lengthwise, with concurrent sacrifice of its marrow and invasion by mucous membrane and submucosal tissue; the head is likewise excavated, the base reduced to a thin plate of either oval or reniform outline.

4. Since the ossicles attain maturity while the remainder of the skeleton is still fetal, the opportunity for structural alteration is great. Since they are remodelled elements—having been profoundly altered to make up an ossicular chain—variation is to be expected, and does regularly occur.

5. These observations must be taken into account in any appraisal of pathological change in the ossicle. Without knowledge of the kind briefly reported here, normal departure from so-called typical form could be erroneously interpreted as the result of disease.

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XL

OSTEOMYELITIS OF THE MAXILLA SECONDARY TO SUPPURATIVE MAXILLARY SINUSITIS

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When a patient presenting signs of osteomyelitis of the facial bones secondary to suppurative sinusitis is encountered, most clinicians rightfully are much concerned about the prognosis. There was always a high mortality rate in this disease before sulfonamides and antibiotics (penicillin) were used as adjuvants in treatment. It has been found difficult accurately to classify clinically the pathologic process in the early stages of the disease, although much has been learned about the pathologic characteristics of the disease. There are two general classes: (1) the localizing or self-limiting type, in which sequestration of bone takes place, and (2) the spreading or nonlocalizing type of infection. It is probable in the light of recent information that the localizing type may be due not only to certain reparative processes in the person afflicted but to a certain degree to the type of infecting organism and to the character of the blood supply of the particular bone that is involved.

In the spreading type of osteomyelitis it is, I think, the general consensus that the nature of the bone and its blood supply are important, but more important is the fact that the infecting organism is an anaerobe as Williams and Heilman[1] have suggested, and therefore is not susceptible to the usual therapeutic measures. It is fortunate that recently adjuvants in treatment have proved effective in combating this type of infection. Studies on the effect of sulfonamides and antibiotics (penicillin) have shown that when they are used in conjunction with well-directed proved clinical and surgical management, the disease may be controlled more satisfactorily than when they are not used. Reliance must not be placed on sulfonamides and the antibiotics alone. It must be emphasized that they are adjuvants in treatment.

A considerable body of literature has accumulated on osteomyelitis of the cranial bone secondary to suppurative disease of the frontal

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sinus. There has been much less written on osteomyelitis of the maxilla secondary to suppurative disease of the maxillary or ethmoidal sinuses.

Disease of the bones of the head was described in the Smith Surgical Papyrus.[2] This report is thought to embrace the period between 5000 and 6000 B. C. According to Wilensky,[3] Hippocrates included a rather accurate description of exfoliation of bone in his "Injuries of the Head." There was apparently a higher incidence of osteomyelitis of the mandible and long bones than of the maxilla and frontal bones.

In 1926, I[4] reported four cases of what I called peridacryocystitis and periosteomyelitis of the maxilla following operation on the maxillary sinus. The symptoms and signs were characteristic. The initial symptom was severe pain in the face developing some days after operation. At this time there was no evidence of swelling or redness but they developed in a few days in the region of the lacrimal sac. Infection in the lacrimal sac could not be demonstrated at this stage. Suppuration took place in the region of the lacrimal sac and drainage was instituted. In one case the lacrimal sac was removed some months later. In two cases infection in the lacrimal sac was never demonstrated. In the fourth case the patient died of meningitis. It was suggested that the complication might have developed because of injury to the nasolacrimal duct when the window of the antrum was fashioned and that this resulted in the involvement by infection of the adjacent periosteum. It had occurred in cases in which intranasal operations had been performed and also in cases in which extranasal or alveolar operations had been performed. In all subsequent cases in which the disease has developed the same clinical syndrome has been observed.

In 1940, Porto[5] gave a report of this disease.

In 1941, Pastore and I[6] reported a case of osteomyelitis of the maxilla following sinusitis. At the time this patient was treated sulfonamides were being used as adjuvants.

In 1941, Tavares[7] reported a case from Brazil. This report was reviewed by Salinger.[8] Tavares collected 11 other cases from the literature. In all cases the termination had been fatal.

In 1945, Hallberg[9] reported two such cases in which penicillin was used.

In 1945, Sadovsky[10] reported a case in which the localizing type of disease was treated successfully.

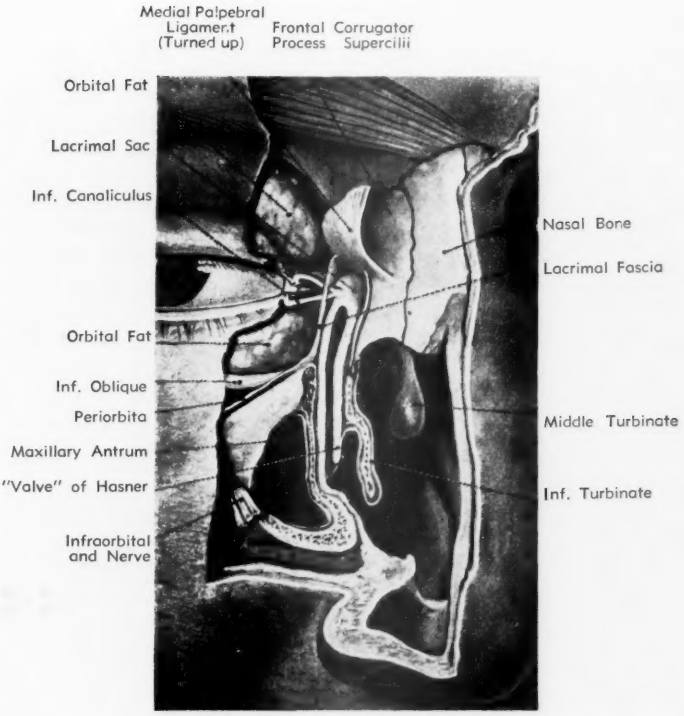

Fig. 1.—Dissection to show the relations of the lacrimal sac and the nasolacrimal duct from in front. (Figure 114 from Wolff[13] used with permission of the publishers.)

The pathologic characteristics of osteomyelitis of the maxilla differ considerably from those of osteomyelitis of the frontal bone. In the first place, the blood supply of the maxilla is derived from terminal branches of the internal maxillary artery. The vessels anastomose freely so that when an infective process involves one of the lesser loops, there is a minimal loss of bone. However, if the main artery is involved, great loss of bone may follow. Havens[11] and Harlowe[12] have made such observations. Because the maxilla does not have porous bone, there is little or no opportunity for pus to be confined within the bone. In the frontal bone, extension of infection takes place by thrombophlebitis of the dural, diploic and pericranial veins, all of which communicate. The venous sinuses in the

diploe play a very important role because infection can be confined in the spaces.

Extension of infection in osteitis or periosteomyelitis of the maxilla must be through the veins in the periosteum for the most part. In the region of the lacrimal sac the direction would be toward the cavernous sinus and the brain through the angular vein.

The anatomic characteristics of the lacrimal duct and the surrounding tissues have much to do with the sequence of events (Fig. 1) which lead to the clinical syndrome. Wolff[13] described the lacrimal sac as enclosed by a portion of the periorbita or lacrimal fascia. Areolar tissue between the sac and the fascia contains a fine plexus of veins continuous with the plexus around the duct except over the fundus of the sac where the sac is closely adherent to the fascia. The medial palpebral ligament is free but it is continued upward and laterally as a sheet blending with the fascia covering the fundus. The portion of the sac below the level of the ligament is covered only by a few muscle fibers. This protection offers little resistance to distention and swelling. It is in the region below the ligament that abscesses and fistulas develop.

The average length of the nasolacrimal duct is 5/8 inch (1.6 cm.). The position and shape of the inferior orifice vary greatly. This fact, it seems to me, is of the greatest importance when considering the syndrome under discussion. In some instances, the orifice is round and corresponds to the opening of the bony canal at the highest part of the inferior meatus. In others the orifice runs as a membranous tube for some distance under the mucous membrane, is found at different points down the lateral wall of the meatus and becomes a slitlike opening as it descends. It may be difficult to identify. The duct is said to have valves but actually the valves are folds of the membrane difficult to identify. The most constant of these is the plica lacrimalis, which is located in the orifice at the lower end. It is said to represent the remains of the fetal septum. The lacrimal duct is surrounded by a rich plexus of veins really forming an erectile tissue resembling the structure of the inferior turbinate. Engorgement of this plexus is enough to constrict the duct at its upper end where the duct can be separated easily from the bone. Below, however, it is closely adherent, forming a mucoperiosteum and thus infection may spread easily from duct to bone or vice versa.

The question immediately arises, should the surgeon in each case identify the lower orifice of the nasolacrimal duct before he

fashions an opening in the naso-antral wall? Could such a refinement in technic be carried out successfully? Would avoidance of injury to the lower orifice prevent the occurrence of osteomyelitis? I am not prepared to answer these questions satisfactorily at this time.

If my idea, that it is injury to the nasolacrimal duct at the time of operation that causes the complication, is correct, then the answer to the questions would be in the affirmative. When one considers the low incidence of the complication and the actual difficulty in locating the lower orifice of the nasolacrimal duct, one hesitates to make positive statements concerning the advisability of locating the lower orifice of the duct.

With the anatomic characteristic of the nasolacrimal duct in mind, the sequence of events leading to the clinical syndrome could be: (1) injury to the orifice of the nasolacrimal duct; (2) congestion of the plexus surrounding the duct, producing severe pain; (3) retrograde thrombosis extending to the lower end of the lacrimal sac, causing swelling of the region of the inner canthus; (4) suppuration, causing abscess in the region of the sac; (5) elevation of the periosteum by an abscess; (6) thrombosis of terminal vessels, resulting in sequestration; or (7) spreading of infection through the angular vessels to the orbit with extension to the meninges, the cavernous sinus or the superior longitudinal sinus, terminating in death.

The cases of osteomyelitis of the maxilla secondary to operations on the maxillary sinus that I have encountered over a period of years always have been approached with misgivings because of the previous unfavorable experience with the disease. More recently the more favorable effect of treatment with sulfonamides and antibiotics (penicillin) in conjunction with other methods of treatment has been so striking that I decided to study the comparative effect of present methods of treatment with those in use before the era of sulfonamides and antibiotics.

Before the use of sulfonamides and antibiotics became known, reliance had to be placed on the use of time-honored methods of treatment: hot compresses and sedatives for relief of pain, surgical drainage of abscesses or phlegmons, transfusions of blood as supportive treatment and subsequent removal of sequestra that prevented final healing. In the localizing type of infections due to staphylococci and hemolytic streptococci, these methods of treatment were reasonably successful. The use of a sulfonamide suitable for the infecting organism proved to be greatly advantageous as an adjuvant.

If the infecting organism proved to be an anaerobe, the effect of treatment with sulfonamides was very disappointing. The ad-

equate use of penicillin under such circumstances has proved to be dramatically effective in certain cases. It must be emphasized, however, that when pus is present sulfonamides and penicillin are not effective until adequate surgical drainage has been established. Further, it should be emphasized that surgical drainage should not be instituted before the formation of an abscess or phlegmon is manifest.

At the present time it would be considered best to start the use of sulfonamides and penicillin at the very onset of the complication. It is possible that in so doing the infection can be controlled before actual suppuration takes place. It is known that both agents are most effective at the time when the organisms are most actively reproductive. If abscesses or phlegmons develop, drainage must be established. Administration of the therapeutic agents should be continued for several days following apparent control of the disease. If sequestration develops, surgical measures designed to remove adequately the diseased bone are indicated. Unlike osteomyelitis of the frontal bone, it is not necessary at the outset to plan a surgical attack intended to remove bone well beyond the diseased bone.

REPORT OF CASES

In this report I am including only the patients I have personally been responsible for. Six other patients have been observed by other members of the staff. The four cases that I reported previously[4] are not included in this report. Cases in which osteomyelitis occurred following injury to the maxilla also are not included. Only those cases in which the disease developed following an operation on the maxillary sinus at the Mayo Clinic or which were referred to me after the development of the complication are reported.

CASE 1—In May 1928, a man 46 years old was seen who complained of severe pain and swelling over the left side of the face and the eyelids. He gave a long history of suppurative sinusitis and stated that he had submitted on May 17 to a Caldwell-Luc operation on the left maxillary sinus. Eight days following the operation severe pain had occurred in the region of the left inner canthus. This had been followed soon by swelling of the lacrimal sac and edema of the eyelids and face.

Examination revealed that drainage had been established in the lower rim of the orbit near the inner canthus. Bone could be felt with a probe. A culture of the pus obtained from the wound re-

vealed staphylococcus aureus. Nothing was disclosed in the roentgenologic examination other than diffuse cloudiness of the region.

During observation in the hospital there was no evidence of any untoward febrile reaction. The examinations of the blood gave results which were considered within normal limits. Hot compresses were applied over the face and after several days an external operation was performed on the maxillary sinus. At this time definite sequestration was encountered and the sequestra were removed.

The condition apparently remained in abeyance for a considerable time. Some months later the patient again presented himself at the Clinic and another sequestrum was encountered and was removed. Since that time he has remained in good health although there is considerable deformity of the left side of the face.

CASE 2.—In 1939 a man 33 years old presented himself at the Clinic. He said that a Caldwell-Luc operation had been performed for a long-standing suppurative infection of the right maxillary sinus. Four days following operation severe pain had developed in the region of the inner canthus. Swelling of the region had followed rapidly and had involved the face and the eyelids. After 12 days an incision had been made in this region but no pus had been obtained.

On admittance to the hospital the patient appeared very ill. The temperature was 103° F. The history revealed that sulfonamide drugs had been used adequately. At least, he was getting 20 grains (1.3 gm.) every four hours. The right eye was closed and there was some swelling of the inner canthus on the left side. There was an incision near the region of the lacrimal sac. The buccal wound was open and draining. No fluctuation could be felt in the edematous soft tissues. Examination of the central nervous system and of the eye gave normal results. The culture of the blood showed no organisms to be present. The culture of the pus from the wound showed a micrococcus to be present. The roentgenologic examination revealed what was said to be osteomyelitis of the floor of the orbit and the maxillary sinus. During the stay in the hospital the level for the sulfonamide drugs was kept at from 13 to 19 mg. per 100 cc. of blood. Two transfusions of blood were given as supportive treatment when it was found that secondary anemia was developing as a result, it was thought, of the use of sulfonamide drugs. Intravenous administration of solution of glucose was used to support the patient further.

On the eighteenth day sequestrectomy was performed with the patient under pentothal sodium anesthesia. Following this there was

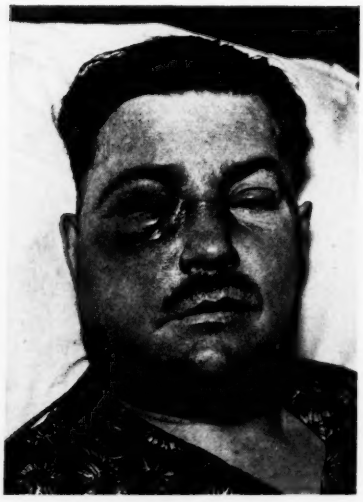

Fig. 2. Case 3.—Patient on entrance to hospital.

marked improvement in the condition and much of the pain that he had complained of during his stay in the hospital was relieved. He was permitted to leave the hospital. He returned on the sixty-ninth day with an acute swelling of the face. Examination revealed a sequestrum presenting in the buccal wound. With the patient under general anesthesia applied through an intratracheal tube this sequestrum was removed and it was found to consist of practically all of the maxilla.

Following this there was rapid healing. The patient has been observed at frequent intervals since. He was last seen in December 1945, and has remained in excellent condition. There is surprisingly little deformity of the face.

Case 3.—A man 43 years old was observed at the Clinic in 1942. He had had maxillary sinusitis for a year and a half following the extraction of a tooth. He stated that in the middle of March 1942 an intranasal antrum window operation had been performed. The immediate convalescence had been without incident but on April 2 very severe pain in the region below the eye and toward the inner canthus had been experienced. Swelling of the eyelids and face rapidly followed. He had been severely ill and the fever had been

high. On April 11 drainage had been instituted and the sulfonamide drugs had been given as adjuvants in treatment.

When the patient presented himself at the Clinic he was seen to be very ill and his case was considered an emergency. The very distressing symptom was that pain was worse at night, occurring in the region of the forehead and the root of the nose. Marked tenderness was elicited over the bregma and in the region of the inner canthus. The eyelids were so swollen that the eye was closed and it was seen that the globe was partially fixed (Fig. 2). The leukocyte count was very high. Examination of the fundus revealed that the disk seemed full. Culture of the discharge from the incision that had been made was found to contain streptococcus viridans. During observation in the hospital great difficulties were encountered with the use of the sulfonamide drugs. The patient did not tolerate adequate dosage.

On May 1 an exploratory type of operation was conducted through an alveolar incision and extensive sequestration involving the floor of the orbit was encountered. This operation did not check the progress of the disease. Swelling occurred over the root of the nose. The clinical course did not seem favorable. A week later a second operation was performed over the root of the nose and the nasal bones were found sequestrated and enveloped in a definite capsule. There seemed to be no direct evidence of involvement of the frontal bone.

The patient's course was unsatisfactory and death occurred on May 28. Necropsy revealed thrombosis of the cavernous and superior longitudinal sinuses and extensive meningitis.

CASE 4.—In 1943 a man 21 years old presented himself at the Clinic with the history of long-standing bilateral suppurative sinusitis. An intranasal operation was performed in September 1943. The immediate convalescence was very satisfactory but on the ninth postoperative day the patient complained of severe pain in the left side of the face, especially localizing it to the region of the inner canthus. At this time there was no redness or swelling. Tenderness was present. Redness and more tenderness occurred after the third day of pain. The pain was not controlled by heat or cold compresses and it was necessary to use sedatives. Swelling developed rapidly and there was severe edema of the lower eyelid. Penicillin was administered intravenously in adequate doses over a period of 25 days. Suppuration developed and drainage was instituted in the region below the inner canthus. Examination of the pus on culture

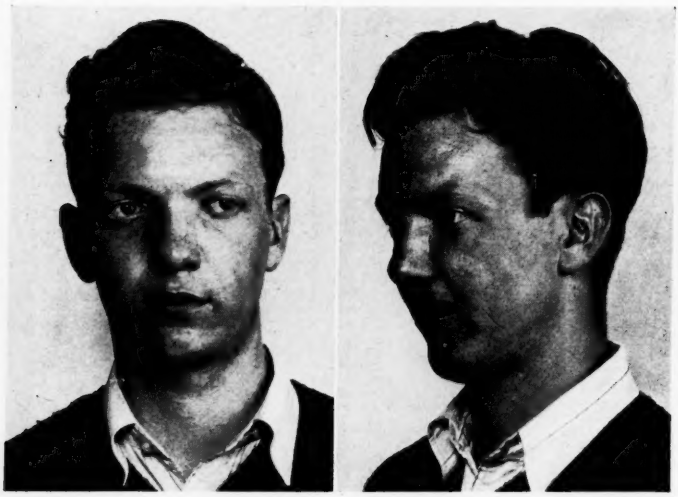

Fig. 3. Case 4.—Patient after recovery.

revealed an anaerobic bacterium. Rapid recovery took place after the drainage of the suppurative process.

The patient was dismissed from obesrvation and returned in February 1944, because the incision at the lateral aspect of the nose had broken down. A small sequestrum could be felt with a probe. In the meantime the patient had gained 25 pounds (11.3 kg.) and considered himself to be in better health than at any time previously. The sequestrum was removed without its being necessary to employ anesthesia. Rapid healing took place and this has remained permanent (Fig. 3). The patient has been seen at intervals of four to six months and has remained in excellent health.

Case 5.—In 1943 a man 36 years old presented himself at the Clinic complaining of long-standing nasal obstruction on the left side. This was found to be due to a wide deflection of the nasal septum and a large naso-antral polyp which filled the nostril behind the deflected septum and presented in the choana.

An intranasal operation for the removal of this obstruction was carried out with an apparently good result and the immediate con-

valescence was without incident. The patient was dismissed eight days after operation.

On the sixteenth day after the operation, the patient complained of pain in the region over the inner canthus. No redness could be seen. Physical therapy in the form of diathermy was used but this was not effective. A rapid swelling of the lid and upper maxillary region ensued and evidence suggesting suppuration was present. Drainage was carried out. The sulfonamide drugs in adequate doses were given over a period of time following the apparent recovery. Penicillin was not used as none was available at that time. The patient was dismissed from the hospital apparently well.

Four weeks following his dismissal a recurrence took place. At this time he was hospitalized and penicillin was administered intravenously for 11 days. Rapid involution of the disease without suppuration took place and he has remained well since. He was last seen in December 1945. There was practically no scarring and no disfigurement of the face. In this particular case sequestration did not take place.

Comment. It is seen that the syndrome developed in a like manner in each case. All patients but one had sequestration. The sequestration involved a considerable portion of the maxilla in three cases. In Case 2 the sequestration was very considerable. In Case 5 sequestration did not develop. It is probable that the use of penicillin prevented thrombophlebitis sufficiently so that the terminal vessels supplying the bone were not involved.

The first case was encountered before sulfonamides were used in the treatment of infection. The infecting organism was identified as staphylococcus aureus.

In Case 2, a sulfonamide was used in what was considered to be adequate dosage but did not change the course of the disease. It may have limited the disease. Examination of the blood and clinical signs suggested that the sulfonamide was not well tolerated. The infecting organism was identified as a micrococcus. Transfusion of blood as supportive treatment had a beneficial effect.

In Case 3, the patient was more seriously ill than is usual. Adequate doses of sulfonamides were used before he came to the Clinic and during his stay in the hospital although some difficulty was encountered in their use. The patient continued to fail despite all supportive treatment. The infecting organism was identified as streptococcus viridans, known to be resistant to both sulfonamides

and penicillin. Penicillin was not available at that time. Death occurred because of extension of the uncontrollable infection.

In Case 4, penicillin was used from the onset and although an abscess developed, the sequestra that formed were relatively insignificant; however, until removed they interfered with healing. The infecting organism was identified as an anaerobe.

In Case 5, penicillin was not available at the initial stage of the disease and reliance was placed on sulfonamide therapy and drainage of a small abscess. Sequestration did not take place. At the onset of recurrence of symptoms and signs, penicillin was administered intravenously for 11 days. The signs disappeared on the third day. Apparently, suppuration was prevented. The infecting organism during the original episode was identified as a micro-aerophilic bacterium.

CONCLUSIONS

1. The mechanism of the occurrence of osteomyelitis of the maxilla following operations on the maxillary sinus is suggested, as the sequence of symptoms and signs seems to be explained on anatomic and pathologic bases.

2. The pathologic characteristics of osteomyelitis of the maxilla that occurs secondarily to suppuration of the maxillary sinus differ greatly from the pathologic characteristics of osteomyelitis of the frontal bone that occurs secondarily to suppuration of the frontal sinus, largely because of the difference in the character of the bone involved and particularly because of the difference in the blood vessel arrangement.

3. The use of penicillin in conjunction with well-known general surgical principles of treatment of suppurative processes is very advantageous and gives promise of great improvement in the management of such a serious disease as osteomyelitis of the maxilla secondary to operation on the maxillary sinus.

4. It should be emphasized that accurate classification of the infecting organism is essential if penicillin as an adjuvant in treatment is to be employed effectively.

5. It should also be emphasized that in the presence of pus neither sulfonamides nor penicillin can be relied on as curative agents. The use of sulfonamides and penicillin should be continued for several days following apparent resolution of the infective process.

MAYO CLINIC.

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XLI

THE DANGER OF UNRECOGNIZED ANOXIA IN LARYNGOLOGY

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While in general the academic and theoretical aspects of oxygen want are well known, it does not seem equally true that the importance and serious implications of anoxia are always appreciated, especially under the stress of emergency. This discussion is intended to show that some degree of anoxia frequently is unsuspected and untreated in conditions which the laryngologist sees or ought to see; to indicate that even brief periods of hypoxia or anoxia may cause serious damage; to point out some conditions responsible for anoxia; to review the signs and symptoms which may indicate its development; and to present measures for its prevention and treatment. Usually, however, it will be sufficient to recognize the condition and diagnose its cause for the right course to be followed.

It has been difficult to establish the true histologic picture of damage from anoxia because the most notable injury is to brain tissue and such changes occur as well with other acute circulatory and degenerative disturbances. Also anoxia is usually associated in man with other grave conditions such as anesthetic depression and septic, degenerative or circulatory disease. In addition it requires a survival time of from 24 to 48 hours for demonstrable changes to develop in brain tissue after injury. Agonal and postmortem changes in this tissue, too, are hard to prevent or estimate.

Windle[1] recently has given a clear-cut demonstration of the effects of oxygen want in guinea pig embryos at term. The uterine circulation was interrupted until asphyxia occurred, the animals were resuscitated, allowed to survive and later killed with the tissue fixed in situ. Although it is accepted that brain tissue of the young is less sensitive than that of adults, all animals in whom the circulation was interrupted for eight minutes or more showed neurologic symptoms and pathologic changes. The symptoms included incoordination, ataxia, spasticity, tremors, convulsive seizures, diminished learning

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ability and even decerebrate states. Histologically, in 65 per cent of the animals there occurred edema, chromatolysis, necrosis, capillary hemorrhages, glial proliferation and loss of brain cells with changes varying from slight atrophy to marked sclerosis.

Morrison[2] has recently demonstrated similar profound changes in the brains of dogs and guinea pigs subjected to repeated short periods of anoxia. The critical level seemed to be a reduction from the normal 20, to 12 or 13 volumes per cent of oxygen in inspired air with repeated exposures of 25 minutes, although for greater degrees of anoxia the time of dangerous exposure seemed to be correspondingly lessened. He found in the monkey that a single exposure to a simulated altitude of 32,000 feet for 25 minutes was capable of producing extensive laminar necrosis of the cortex.

Frank necrosis occurred usually only after episodes of anoxia sufficiently severe to produce cessation of respiration. The frontal lobe was most often involved, the temporal lobe least often. The cerebellum was more frequently involved than the basal ganglia and the medulla least. These experiments and many others[3, 4] from the laboratory show definitely the grave and lasting effects of even short periods of anoxia of as little as three to five minutes.

Yet these do probably not represent the extreme picture since it is very difficult to exclude such reservoirs of oxygen supply as the placenta and the lungs, as in Windle's experiments, or to completely exclude cerebral circulation through collaterals even after ligation of the carotid and vertebral arteries.

In clinical conditions, it has been somewhat difficult to establish the picture of anoxia. The studies by Courville[5] of material obtained after death from nitrous oxide anesthesia are classical and his conclusions that these represent the effects of anoxia are quite generally accepted. He lists such changes as sclerosis of scattered pyramidal cells, patchy necrosis, degeneration of limited portions of the cortex and lesions in the lenticular nucleus. Degenerated areas tend to become confluent and to bring about necrosis of the various laminae. It is to be remembered that only when the patient survived a sufficient time, usually 24 to 48 hours, are histological changes found, and in the more fulminating cases these were not demonstrable. He believes irreversible changes may occur after three to eight minutes.

Many authors have shown similar changes, as for instance Schreiber[6] in asphyxia neonatorum, Hartman[7] in surgical conditions. It is not beyond controversy that the effects of carbon monoxide

poisoning are due solely to oxygen want so the many reports on this disease are not cited.

The data so far presented are related to gross laboratory and clinical demonstrations. But it is hardly to be doubted that in patients surviving, there are important changes besides those effecting immediate circulatory collapse and general depression. Courville[8] lists among sequelae of oxygen want, twitchings, convulsions, even decerebrate rigidity, residuals of parkinsonism, emotional flattening or psychic aberrations. He believes that the condition of many patients in hospitals for the feeble-minded or the insane should be classed as postanesthetic states due to anoxia.

MacClure[9] reports the case of a brilliant girl who had led her college class, but after nitrous oxide anesthesia for dental extraction, was forced to leave school because she could not keep up in her school work. I have seen rather marked emotional changes after bulbar poliomyelitis in patients who suffered prolonged periods of anoxia.

Thorner and Lewy[10] believed that even one single period of anoxia of 60 seconds' duration leaves distinct evidence of morphologic changes and that no prolonged period of oxygen want will leave the brain totally unscathed.

Barach[11] concludes that anoxia in nitrous oxide anesthesia has produced death from anoxia, psychosis from permanent brain damage, personality changes, and impairment of circulatory and respiratory function which may contribute to atelectasis, edema or cardiac failure. (He does not, however, condemn this anesthetic if properly given.)

An editorial in the Journal of the American Medical Association states that asphyxia of even short duration must be regarded as "a grave hazard to the great majority of persons."[12]

It does not seem necessary to go further into the great mass of available evidence to prove that oxygen want even for short periods and perhaps of mild degree may be serious. I shall attempt, however, to point out some of the situations, frequently unsuspected, in which it may devolve upon the laryngologist to recognize the factor of anoxia and to aid in its correction before irreversible changes have taken place.

Anoxia is usually classified after Barcroft and Peters and Van Slyke,[13] as follows:

1. Anoxic anoxia—resulting from defective oxygenation of the blood in the lungs due to (a) low oxygen in the inspired air, as

at high altitude, (b) pulmonary abnormality, as obstruction, pneumonia, edema, atelectasis.

2. Anemic anoxia—resulting from insufficient means of oxygen transport as in anemia or carbon monoxide poisoning.

3. Stagnant anoxia, in which there is poor delivery although the hemoglobin may be saturated, due to heart failure, obstruction of venous return and shock.

4. Histotoxic anoxia—resulting from the cells' inability to utilize oxygen, as in cyanide poisoning.

We are concerned chiefly with the anoxic type as it is related to abnormalities of the respiratory tract.

The symptoms of anoxia are important to have in mind, especially in dealing with conditions associated with similar manifestations. Close analysis will disclose that in many of these it is the anoxia itself which causes the disturbances which may have been ascribed to sepsis, decompensation, shock, virus infection and the like.

Mental symptoms are early. As Barcroft points out, these may resemble those of acute alcoholism and may vary with the patient's temperament and surroundings. There may even be preliminary exhilaration and excessive confidence in faulty judgments, such as aviators have at high altitude. More common, however, are depression, confusion, disorientation, irrationality, unresponsiveness, lethargy and finally coma. Antagonism and combativeness occur often. This was found in a considerable percentage of my bulbar poliomyelitis cases and has been important because it has made necessary more active measures, such as tracheotomy, which could have been avoided if the patient had cooperated in simple aspiration. It was interesting in reviewing the nurses' records of bulbar poliomyelitis cases to find that confusion and irritability had been frequently noted before respiratory difficulty was yet suspected.

Restlessness is an important early symptom both because the active patient increases his oxygen want and also because the unwary are too often tempted to attempt its relief by sedation which may be followed by complete surrender by an overburdened respiratory mechanism.

Dyspnea is usually an early symptom but may not be recognized easily in an exhausted patient or in one who has had sedatives.

Cyanosis may be a late symptom of oxygen need since it may not occur while there is compensation by increased rate or depth of

respiration, and increased rate and stroke output of the heart. If obstruction develops slowly, cyanosis may be a late sign and I believe that if severe cyanosis develops in acute tracheobronchitis of less acute onset, we are liable to have overwaited the indication for tracheotomy. Cyanosis may be absent in anemia since it depends on an absolute amount of reduced hemoglobin in the capillaries. Likewise, it may not appear in the presence of carbon dioxide deficit, in which condition surface capillaries are constricted. Such a condition may occur after shallow breathing in encephalitis, and possibly where there is much fluid in the tracheobronchial tree. This may occur in the type of tracheobronchitis described by Gittins[14] which may have only a gray cyanosis and few obstructive signs. This may be, however, simply part of a shock picture.

The effect of anoxia upon the heart is apparently not so great as that of the usually associated carbon dioxide excess.[15] There will be frequently an early increase in rate and stroke output with later rise and finally serious disturbance of rhythm. It is to be remembered that a rapid pulse of poor quality is a sign of imminent heart failure which again may mean that we have waited too long to intervene.

Bulbar poliomyelitis was one of the conditions which rather rudely awakened me to the need of alertness in considering anoxia. Perfunctory contact had led me to believe that the effects were, as seems generally accepted, chiefly due to localization of the virus in specific nerve nuclei. A group of interns had been rather frantically trying to keep alive a patient with proven poliomyelitis who had gone from confusion and disorientation to complete coma. An alert supervisory nurse suggested that a laryngologist be called. On seeing this patient, my first impression was that his depression was due to virus infection or sepsis. As described in a previous report,[16] direct laryngoscopy disclosed that the pharynx, larynx and trachea were filled with secretion and aspiration produced improvement for a time. Eventually respiration ceased completely for an estimated three minutes while a tracheotomy was done. Secretions were aspirated, and artificial respiration was given until the patient could be placed in a respirator. He was conscious in 45 minutes and nearly normal mentally in two hours. It is hard to see how any other condition than anoxia could have produced a condition so quickly correctable.

In a study of quite a number of cases of bulbar poliomyelitis in which the primary disturbance has been in the swallowing mechanism with the flooding of the airway, it has seemed to me that anoxia has

been the chief trouble. Even the disturbed rhythm, which was held to be part of the central picture and was considered to make these patients unsatisfactory candidates for the respirator, seemed due to anoxic depression of the center combined with instinctive or reflex resistance to drowning from the secretions. On clearing the airway of these patients most of them developed a fairly good rhythm and did well in the respirator.

The reverse of the preceding experience occurred because I did not myself appreciate the seriousness of even limited periods of hypoxia. A young woman with bulbar poliomyelitis was doing well as long as the secretions were aspirated. She was found, however, during a nursing shortage under the care of a relief student nurse, badly blocked with secretion, cyanotic and unconscious, this condition having occurred within a half hour. Aside from correcting the immediate condition nothing further was done, as I had been criticized for suggesting tracheotomy in such cases with the statement which is probably true, that careful nursing, postural drainage and aspiration should make it unnecessary. In this patient another half hour of serious interference with oxygen intake occurred due to fluid accumulation. The patient sank into a profound coma with dilated pupils and lost reflexes, apparently a true decerebrate state from which she did not recover, although in the respirator her heart continued to beat for two days longer.

This and several experiences brought me back to my original belief that when the patient with bulbar poliomyelitis is not doing well because of inability to get proper care, or uncooperativeness which may itself be a result of anoxia, tracheotomy may be a life-saving measure.

Any condition in itself depressing may make a patient unduly sensitive to slight degrees of oxygen want and such patients must be closely watched. Shock is likely to have associated with it a stagnant anoxia due to pooling of the blood in the splanchnic area.[17] Not only may oxygen administration then be of value but it is important that the airway be kept clear and the patient, especially if unconscious, be placed in the best position to breathe easily. Military surgeons report that unconscious, shocked casualties were frequently greatly helped by turning the patients to the prone position with the face to the side.

A case in point was a young woman who had been shot through the frontal lobes with explosive effect and had been unconscious for eight days. The nurse reported that she was dying. She was found

with a thready pulse and gasping shallow respiration, lying on her back with sagging jaw and tongue. Although she was in an apparently terminal state, slight retraction suggested respiratory obstruction. A bronchoscope was hastily passed, considerable thick mucus aspirated, oxygen given, the patient placed in the half prone position and she lived to become a fairly normal individual.

I have written of anoxia in tracheobronchitis and respiratory obstruction considered chiefly in terms of terminal asphyxia. It should rather have been pictured like a wolf at the heels of a wounded animal, nagging, harrying and tiring until it closes in on its weary prey in one final cruel slashing attack. From the moment in respiratory obstruction that any degree of oxygen want develops, it begins to operate adversely and unless relieved it may be largely responsible for an unfavorable outcome. It produces restlessness with further activity and increased oxygen consumption. It and its accompanying carbon dioxide accumulation lead to increased respiratory effort, increased negative thoracic pressure, increased congestion, exudate and even capillary hemorrhage, increased lymph flow and increased capillary permeability, shown by Drinker[18] to follow anoxia. This exudate leads to still further blocking of the bronchioles and the lung bed, with perhaps atelectasis and emphysema and deepening oxygen want. There is evidence also that vasomotor tone is seriously impaired by depression of function of the suprarenal glands by anoxia.

By now the respiratory center is depressed, the heart and the respiratory muscle cease to function efficiently due to oxygen need and the fight nears its end. In this as in many other conditions associated with anoxia, the physio-pathology must be visualized almost from the outset if it is not to become irreversible or leave tragic sequelae. Oxygen administration and other measures will relieve most cases of tracheobronchitis and respiratory obstruction but no undue complacency should, in progressive cases, delay the certain and relatively safe relief by tracheotomy.

Anesthetic misadventures are perhaps the field where anoxia plays the greatest role and Courville[5] believes that accepted figures of mortality and morbidity for nitrous oxide are wrong and he has reported nine deaths and other serious accidents he believed due to anoxia from this agent. He[19] has reported also serious sequelae from ether which he considers the simplest and safest of anesthetics. Waters'[20] recent statistics, although they do not separate anesthetic deaths from other operative and immediate postoperative causes, suggest a much higher figure for such accidents than those usually given.

The modern trained anesthesiologist is alive to the hazards of anoxia and has given much thought to the problem and its prophylaxis, so that it would be presumptious to discuss at any length this phase of the subject.

A question may be raised, however, whether there may not at times be undue confidence in procedures which, though having much greater potentialities, must yet be used with greater alertness and discrimination. The normal respiratory apparatus with its accurate nervous, chemical and reflex control is not likely to have a perfect mechanical substitute.

Some matters for serious consideration are: the heavy levels of premedication with morphine, scopolamine and barbiturates often advised before general anesthesia; the use of curare, unless quite essential as in some chest surgery, with its paralysis of the voluntary muscles and a loss of some of the excursion desirable for good circulation in the lung bed necesary to prevent congestion and even atelectasis; the more or less irregular introduction, in fairly large doses, of irremovable intravenous agents with laryngeal spasm not always perfectly counteracted; the dependence at times on intratracheal insufflation of oxygen without chest movement which Comroe and Dripps[21] believe seriously dangerous for longer than 15 minutes. I am not yet ready to forget the teachings of Chevalier Jackson and St. Clair Thomson, that one must not dull for long the cough and other protective mechanisms of involved air passages by opiates or other drugs, though some anesthesiologists consider us alarmists in this situation. The laryngologist in his own cases at least can not delegate entire responsibility and must be alive to the hazards of anoxia in anesthesia and know the counter measures. If he works in one of the smaller hospitals where he assumes some of the responsibility, he may even gain some satisfaction in cooperating with the efficient and pliable nurse anesthetist.

Especially since laryngological procedures usually involve the airway it is his responsibility to see that it is clear during anesthesia. In a patient on the dangerous edge of anoxia the additional block of the tongue clumsily pressed into the pharynx or the accumulation of blood or secretions may be very dangerous.

One very interesting problem is the relationship of anoxia to possible carbon dioxide deficit. In spite of the strong belief of Haldane and others that this may be frequent there is still much disagreement of physiologists on this subject. Grodins and Adler's[22] experiments indicate to them that there is no deficiency in chemical stimuli to the respiratory center "during the entire course of obstructive asphyxia."

Men like Barach[23] with considerable experience in inhalation therapy believe that carbon dioxide should seldom be given in such cases.

Excessive elimination of carbon dioxide has been shown to occur after forced breathing. Best and Taylor state that in certain types of shallow breathing as in pneumonia, after breathing of irritant gases, occasionally in encephalitis and even in severe anoxia itself, by its depression of the respiratory center, there may be dangerous loss of carbon dioxide. Thereby, they state there may be induced "one of the gravest types of anoxia since the cardiovascular system as well as suffering from oxygen want is seriously affected by the excessive loss of carbon dioxide . . . The acapnia would also magnify the oxygen want of the tissues for the hemoglobin gives up its oxygen less readily at low oxygen tensions."

Negus[24] has reported a dramatic instance of carbon dioxide need in his discussion of sudden death after successful tracheotomy. His explanation was that after tracheotomy in a previously anoxic patient with increased respiration the free carbon dioxide is quickly blown off. "The acid base ratio is upset, the percentage of carbon dioxide present as carbonic acid is suddenly lowered, while that fixed as carbonate remains stationary. The arterial blood has become so alkaline that the respiratory center no longer receives the necessary stimulation," and respiration ceases. He advised, to prevent this, very slow opening of the trachea or administration of carbon dioxide. Some physiologists believe, however, that the respiratory failure occurs because the respiratory reflex had been previously maintained not by the action of carbon dioxide on the medullary center, now not functioning properly, but by the effect of anoxia on the chemoreceptors in the carotid area—and that this stimulation is gone with sudden access of oxygen. Seevers calls a similar condition developing after high carbon dioxide levels during anesthesia an uncompensated respiratory alkalosis. He states that it takes as long for readjustment when the patient returns to breathing air as it did to adjust originally to the high carbon dioxide atmosphere.

A marked reduction of the carbon dioxide content of the venous blood might be an indication of this state. An improvement as judged by clinical signs and the patient's feeling of relief on cautious administration for a short time of 5% carbon dioxide would be another sign. This could more accurately be determined by finding of the pH and the carbon dioxide content of the arterial blood. According to Gamble,[25] knowing that the disturbance was respiratory rather than metabolic, the relative carbon dioxide excess or deficit could then be closely estimated.

It has been the intent of this discussion to make the reader visualize the narrow margin between oxygen want and disaster. It is hoped that it will help save some anesthetized patient on the edge of dangerous anoxia from being throttled with sponge or instrument; or a sufferer from bulbar poliomyelitis from drowning in his own secretions; or another from struggling for air with a lung bed not functioning properly when that condition can be relieved. And may the picture be so clear that even in states of shock, exhaustion, cardiac failure or the like where stagnant anoxia has already crippled the brain and the heart, there will not be permitted the further shutting off of precious oxygen by poor position or obstructed airway.

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XLII

INTRANASAL MEDICATION

AN INVESTIGATION INTO THE RELATIVE MERITS OF CERTAIN METHODS OF INTRODUCING FLUIDS INTO THE NOSE

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The introduction of fluid medicaments into the nose has always been a favourite form of local treatment for most nasal disorders.

In order that such medicaments may be allowed to act to the fullest advantage, it is clearly desirable that they should come into contact for an adequate period with as wide an area of the affected nasal mucous membrane as conditions will permit.

The upright position adopted by man, and the configuration of the outer wall of the nasal fossa, both hinder the even distribution of fluid instilled into the nose by the conventional methods of drops, sprays and sniffs. In order to overcome the disadvantage of having to diffuse the fluid against the force of gravity, Proetz (1927) advocated the head-low position, and later Parkinson (1933) described a head-lateral position. Recently mention has been made of a forward head-low position, which is said to be effective without being uncomfortable.

An inquiry (Lancet, 1914) into the claims made for the value of the nasal instillation of a solution of Penicillin Patulum (Lancet, 1933) in the treatment of the common cold made it necessary, for the purposes of the inquiry, to decide upon a standard method of introducing the solution into the nose. It was felt that in order to give the preparation, which was known as Patulin, a fair trial it was important to employ a method that brought the solution into adequate contact with the widest possible area of nasal mucous membrane.

Before advising on the method of intranasal medication to be employed, I carried out a series of tests with different methods in a small series of normal subjects. Later these tests were repeated in another small group of normal subjects with almost identical results.

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In order to test the efficiency of the methods under review, it was decided to take advantage of the paralytic effect of cocaine upon sensory nerve endings. It was known that the application of a solution of cocaine to the area of the nose supplied by the trigeminal nerve rendered it insensitive to common sensation, and it was considered that in addition it probably abolished the chemical sensation stimulated by pungent fumes, such as ammonia, chlorine, and menthol. At the time of this investigation no information could be found concerning the effect of cocaine on the olfactory nerve endings, but it was hoped that its application in a suitable concentration to the olfactory mucous membrane would paralyse the sense of smell, in the same way as it is known to affect taste. Preliminary tests confirmed this hope, and it has since been found that the paralytic effect of cocaine on the olfactory nerve endings had already been noted by Zwaardemaker.

It was decided, therefore, to compare the effect upon olfaction and the chemical sense of introducing in different ways a solution of cocaine into the nasal passages of normal subjects.

The number of methods to be tested was limited to the following four, all of which are in general use for intranasal medication, though of course many different variations of each method are practised.

1. *Drops.* With the subject seated in an ordinary wooden chair of the "Windsor" type, the head was inclined as far backwards as was compatible with comfort and one dram of the cocaine solution was introduced by a medicine dropper, drop by drop, into each nostril, the subject being instructed to breathe through the mouth. In all cases a pause was necessary after the solution had been introduced into each side, because it rapidly dropped into the throat and had to be spat out to prevent its being swallowed.

2. *Spray.* With the subject sitting upright, a trained observer sprayed, under direct vision, each side of the nose in turn with an all-glass spray. At first the spray used was so fine that only half a dram could be conveniently used on each side, but later a coarser spray was used so that one dram of the solution could be used on each side. The results were the same with both quantities. With each spray the subject was instructed to inspire through the nose.

3. *Sniff.* With the head bent forward, one dram of the solution was sniffed up each nostril in turn.

4. *Head-Low Position.* With the subject lying supine with the head hanging backwards and downwards so that the crown of

the head faced the floor, one dram of the solution was dropped by a medicine dropper into each nostril in turn, the subject breathing through the mouth. This position was maintained for 30 seconds after the solution had all been instilled.

After some preliminary trials with solutions of different strengths, it was decided to carry out the tests with a 10% solution of cocaine.

Because of the paralytic nature of the solution it was necessary, and on account of its potency it was felt desirable, to carry out only one test on any subject in any one day.

The following tests of function were started not more than two minutes after the end of each test instillation.

Olfaction. Solutions in light liquid paraffin of nitrobenzol (3.05 gm. per liter), guaiacol (0.302 gm. per liter) and xylol (1.75 gm. per liter) were prepared and employed according to the method described by Proetz (1941), and it will be noted that the concentrations used were 100 times stronger than the minimal perceptible odour which he established for each substance. In addition a bottle of plain liquid paraffin was also used to check, from time to time, the accuracy of the responses.

Before testing, the subjects were allowed to make themselves familiar with each of the odours and at each test identification of the odour in question was requested. Three types of reply were noted.

(a) The odour could be identified. This was considered normal and recorded as "unaffected."

(b) The odour could not be identified, but an aroma could be appreciated. This was considered to be a diminished response and was recorded as "diminished."

(c) No odour could be detected. This was considered an abolition of olfactory function and recorded as "abolished."

Chemical Sense. The sensitivity to pungent vapours of that part of the nasal mucous membrane supplied by the trigeminal nerve was tested for its reaction to the inhalation of ammonia fumes. In the unanesthetised nose it was found that quite a gentle sniff from a two-ounce bottle of Liquor Ammoniae Fortis held within two inches of the nose was sufficient to cause a painful reaction with subsequent lachrymation.

The reactions to testing with ammonia after intranasal instillation of the cocaine solution were also grouped under three headings.

(a) No deviation from the normal response was either observed or experienced. This was recorded as "unaffected."

(b) There was an increased toleration of the ammonia fumes with a noticeable diminution in the local sensation and the lachrymation. This was recorded as "diminished."

(c) There was no sensation in the nose nor was there any lachrymation. This was recorded as "abolished."

SUMMARY OF RESULTS OF TESTS OF OLFACTION AND CHEMICAL SENSATION

METHOD OF INSTILLATION	SENSE	EFFECT		
		UNAFFECTED	DIMINISHED	ABOLISHED
Drops	Olfaction	7	0	0
	Chemical sense	5	2	0
Sprays	Olfaction	4	3	0
	Chemical sense	1	5	1
Sniffs	Olfaction	3	4	0
	Chemical sense	2	5	0
Head-low position	Olfaction	0	1	6
	Chemical sense	0	2	5

It was felt that the results were sufficiently uniform to justify drawing certain preliminary conclusions from this small series of cases, and these will be summarized under each method.

Drops do not appear to reach the higher part of the nasal fossae at all and only in two cases was there a slight effect upon the respiratory area. It would seem that by this method of instilling fluid into the nose only a very small area of the nasal mucous membrane is affected, and it does not appear to have enough time in which to act effectively upon the membrane with which it comes into contact.

Sprayed solutions affected the olfactory area in three cases only, and then but slightly. The respiratory area was affected in five cases and in one of these it was paralysed. Vigorous spraying under good conditions of direct vision enabled the respiratory area to be reached in most cases, but here again the solution did not often remain in contact with the mucous membrane long enough to produce its maximum effect.

Sniffs or douches affected the olfactory area in four cases, but in no instance was olfaction abolished.

The respiratory area was affected in five cases but paralysed in none.

By this method it appeared that a wider area of the nasal mucosa could be reached than by drops or sprays, but once again the inhaled fluid did not have time to act effectively.

In the head-low position olfaction was affected in all cases, being abolished in six cases and diminished in one.

The respiratory area was affected in two cases and sensation was abolished in five cases.

The striking difference in the results between this method and the others can, it is suggested, be attributed to two main factors. In the first place the fluid, encouraged by gravity, filled the nose, thus coming into contact with the whole of the accessible nasal mucous membrane; and in the second place, it was kept in contact with the membrane sufficiently long to have an effective action.

This small investigation could, with advantage, have been extended both in scope and in the number of subjects tested, but the results seemed to be sufficiently conclusive to answer the purpose for which the investigation was started. Moreover, the subjects of the test, with one exception all young adults between the ages of 20 and 30, found the effect of applying solutions of cocaine to their noses on at least eight different occasions, definitely unpleasant. I can speak with personal experience of this investigation, because I was one of the subjects, and the discomfort experienced makes it difficult to appreciate why the application of cocaine to the nose in physically healthy subjects could ever lead to an addiction. It was noted that sensation gradually returned, until, within four hours at the most, it appeared to be normal.

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XLIII

APHONIA

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Aphonia is defined as loss of voice, but it is loss of voice which is not due to a central lesion. This discussion will be limited to functional aphonia, or perhaps a better term would be psychosomatic aphonia. This type of aphonia has been, and is, more commonly referred to as hysterical aphonia. If the term hysterical aphonia is used as the name of the disease, it will be well to set up a basis whereby a better understanding of this syndrome can be had.

It is necessary to examine the etiological factors in hysterical aphonia. It is well to discuss the psychosomatic conception of hysteria, of which aphonia is one of the multitude of symptoms, hysteria being a mental disorder which is always due to emotional causes and not to organic causes.

The manifestations of hysteria are myriad, and almost any organ, or organ system, may be the site of these manifestations. When the basic mechanism in hysteria is analyzed, some type of conversion is most commonly found. By this it is meant that an emotional problem is converted into a physical manifestation. The individual is faced with a problem for which he is not able to find a satisfactory, efficient, and acceptable solution at the moment. It may be that the solution of the problem, if the individual acts in accordance with recognized principles of social behavior, is distasteful, unpleasant, or actually dangerous. For this reason he hesitates to do the difficult or unpleasant act demanded by the realities of the situation, but he finds every other pathway of escape blocked because of criticism by society or by his own conscience.

A simple example of this is the soldier who is ordered to advance to a dangerous position which is defended by enemy machine guns. He is forced to advance by his own conscience, by his concept of bravery and soldierly behavior, by his own self-esteem and the con-

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viction that his comrades will hold him in disdain if he does not comply with the order. On the other hand he is held back by the fear of pain, mutilation or death which almost certainly will occur if he follows the order to advance. He is thus caught between the ethical concepts of courage, obedience to orders, and service to society on one hand, and his own desires of self-preservation on the other hand. The psychological and emotional conflicts become severe, and it may be that he is unable to force himself either to advance or to refrain from advancing. In this instance the psychological dilemma is resolved by the conversion of the conflict into physical symptoms; that is, paralysis of the legs, hysterical blindness, or convulsions. Any one of these conversion manifestations is sufficient to enable him to escape from the distasteful situation without loss of prestige or personal esteem.

In ordinary life many individuals will be caught in a similar conflict, and they may not be able to cope with the situation at the moment. This emotional conflict will bring about a marked emotional tension and intolerable anxiety. When this conflict becomes intolerable, the process of conversion with the production of some physical symptom is both a symbolization of, and a solution to, the conflict which has been raging. The question then arises as to which persons will adopt this mode of solving the psychological dilemma.

Psychiatrists have found that individuals who have been badly spoiled in childhood, individuals who are emotionally immature and fundamentally childish in their attitude toward life, and similar types of self-indulgent individuals will often utilize this method of escape. It is frequently found among persons of a rather primitive and somewhat childish make-up, but on the other hand it has been noted among better integrated personality types under tremendous emotional stress, such as war, sudden catastrophe, train wrecks and other highly emotional experiences. More often, however, it is found in individuals who seem to have an insufficient ability to meet the ordinary vicissitudes of modern life.

I have mentioned before that the physical symptom is a symbolization of, and a solution to, the conflict. This means that the physical symptom is not chosen at random, but that an unconscious selection has occurred and the physical symptom is carefully chosen to fit the problem. A symptom is chosen which will evade the issue at hand, such as a paralysis of the limbs, or a severe limp, as in the case of the soldier who had been ordered to march in the front line. It is also necessary that the symptom chosen will solve the conflict

in which the patient is placed. In the example of the soldier the paralysis of the leg symbolizes his conflict over "walking to his death" and at the same time removes the problem, because a paralyzed soldier obviously cannot march to the front. It must be emphasized, however, that this conversion and the production of physical symptoms occur unconsciously and that the patient is not aware of the nature of his symptom or of the cause. He finds himself possessing a physical symptom and does not realize why he has developed this symptom or what it represents, but instead he firmly believes he is suffering from some organic catastrophe. The unconscious and unwitting development of the symptom by this hysterical patient differentiates him from the malingerer who willfully and purposely simulates his symptom for the purpose of deception to gain his own ends.

Hysterical, or conversion, aphonia represents a specialized form of the previously mentioned mechanism. In this instance the individual is unable to talk in a normal voice, or, if able to say a few words, his voice soon dies away to a whisper. There are numerous tests which otolaryngologists use to detect whether or not the presenting symptoms are organic or not, and they need not be enumerated here. If the total situation of the patient is investigated carefully, it will be found that the hysterical, or conversion, aphonia in some way symbolizes the problem and offers a solution to it. For example, a patient who consulted me with this type of aphonia was dissatisfied with his lot in life, his marriage, and his vocation. It was his obligation to go to the employer of a coal mine and ask for a job, but he whispered almost triumphantly to his family, "No one would hire a man to work in the mine who cannot talk out loud to ask for a job, let alone talk and yell to the other miners." He found that his aphonia would not enable him to ask for work, and it would actually prevent him from working if he had a job.

A middle-aged woman whispered to me of her antagonism and hatred for her mother-in-law who lived with her and who, in spite of the fact that she was very hard of hearing, wanted to carry on a conversation all the time. Conversation with the mother-in-law required shouting into an ear trumpet, and this usually brought impatient, sarcastic or resentful rejoinders. The patient smiled happily as she whispered that she had lost her voice. She had tried to be a good daughter-in-law, and certainly no one could blame her if she did not converse with the old lady all day long.

Mrs. M. consulted me for loss of voice. She had been ill for several years having had many complaints. She had had a functional aphonia for three years. After a tedious and painstaking conference with her she finally admitted that by whispering she received a great deal of sympathy which she had never received before. She was induced to speak normally before leaving the consultation room. She was then referred to a psychiatrist for further handling of her case, and, as far as I know, she has never had a recurrence.

Mr. C. R., a farmer, consulted me for loss of voice. In the history of his case it was discovered that he was worried because his son was about to leave home to take up residence in another state. After the patient's voice was gone, except for a whisper, his family sympathized with him and his son remained at home. On gaining his confidence he admitted to me that this was true, and by persuasion I convinced him that he could talk. Before leaving the consultation room his voice was normal, and as far as I know, it has remained so ever since.

Miss M., the daughter of the president of a normal school, consulted me for the loss of voice which she had had over a period of three months. It was found that her parents had forbidden her to see a certain boy friend of whom she was very fond. She arose one morning and could speak only in a whisper. This patient was treated by using an astringent to her larynx. She uttered a considerable sound and was told that inasmuch as she had made a sound she could talk, which she did. After two weeks she had a recurrence, the same procedure was carried out and she recovered her voice, and four weeks later she had another recurrence with the same treatment and the same result. It was then decided by the family to allow the girl to see her boy friend at weekly intervals, and after that there was no recurrence of the whisper.

Mrs. D. could speak only in a whisper for three years, except for a few short intervals when she had some local treatments to her larynx. For a short time she would have a normal voice, but soon would relapse into the whisper. It was discovered that her stepson, whom she disliked, lived with the family. Upon further questioning it was found that when she did not talk, a great deal of sympathy was expressed toward her, even by the stepson, but when she had her normal voice she received no sympathy. It was decided to have the stepson reside with other relatives. She was persuaded that she could talk normally and she did so without any local treatment. She turned out to be a psychiatric problem and was referred to a psychi-

atrist for observation and study. To my knowledge this patient never has had a recurrence.

Mrs. V. H. had a loss of voice for a period of one month. In discussing the condition with her I thought she was holding something back and I asked her whether she had had a familial disturbance of any kind. She burst into tears and told me that she thought her husband was paying attention to another woman and since learning of it she had lost her voice. I asked her to have her husband come in to see me. During my interview with him I learned that he was not philandering, whereupon I asked them to come in to see me together. Mrs. H. admitted that since she had lost her voice her husband had, she thought, been more attentive to her; therefore, she had continued to talk in a whisper. After convincing her that she was wrong in her surmise as to her husband, and that I was sure she could speak in a normal tone, she did so, and continued doing so. I have seen this patient from time to time and she seems to be happy.

Mrs. J. A. had been unable to talk above a whisper for four months when she consulted me. She had had this condition three times previously, each attack lasting from three to five days. She gave a history of having had an appendectomy, some type of ovarian operation, pyelitis which was worse during pregnancy, constipation, anosmia and many other minor illnesses. She was a poor sleeper and was very neurotic. I could not elicit a history of a familial disturbance of any kind. She did state that her family and friends were sympathetic with her in the loss of her voice. I told her I was sure they would be equally happy if she were able to talk and she agreed that they would. I worked patiently with her but could not get a sound by any suggestion that I could offer. I finally resorted to a local treatment of tincture of chloride of iron to her larynx. When this was applied, she made a very noticeable sound, and I proceeded to follow this up by asking her to utter words. Before she left the office, she was talking and has continued doing so to the present time. I feel, however, that if I had unearthed the real cause I could have induced her to speak by treating the case as an organic condition.

In each of these instances the aphonia was merely the presenting symptom of a major emotional disturbance and represented the only avenue of escape from a difficult dilemma. In this sense the larynx became the organ of conversion of the psychological problem and was not itself the site of a pathological process.

This discussion will serve to indicate the therapeutic necessities in the handling of such patients. The otolaryngologist would not

treat a patient for the general symptom of "cough" without ascertaining the cause of the cough and the basic underlying pathological disturbance, and then attempting to eradicate it in its entirety. Treatment directed only toward the aphonia may be successful in restoring the voice by direct and often primitive attacks upon the larynx. The removal of the symptom, however, leaves the patient with unsolved conflicts, and these conflicts will soon be converted into symptoms elsewhere in the body. Thus the aphonia becomes converted into backache, anesthesia of an arm or any of the other hysterical phenomena.

The treatment of functional, or conversion, aphonia must be directed toward the basic emotional problem. A direct attack upon the larynx with astringent drugs, galvanic current, or other suggested measures may bring temporary amelioration but may cause the symptom to return at a later date or in a different form. The patient is no longer open to an attack upon the emotional problem because the otolaryngologist has, by his therapeutic procedures, tacitly implied that a laryngeal disturbance which requires organic treatment does actually exist. There can be no second chance then for emotional treatment because the patient who is anxious to hold on to his symptom then attacks the doctor with, "But if there was no disease in my throat, why did you treat it?"

The treatment of these cases of aphonia can often be carried out by an experienced, sympathetic and understanding otolaryngologist who is willing to listen to the patient's story and to help him understand the underlying factors which brought about his illness. It is only by these means, and in difficult cases by calling in a competent psychiatrist, that the otolaryngologist is able to reach the ideal method of treatment: treatment of the patient as a whole and in such a way to restore health, happiness and efficiency to the total organism rather than to remove the symptoms from a single organ.

SUMMARY

1. Aphonia is defined as loss of voice, but it is loss of voice which is not due to a central lesion.

2. This type of aphonia may well be called conversion aphonia because an emotional problem is converted into a physical manifestation.

3. This type of aphonia is a symptom, and as such should not be treated locally.

4. An attempt should be made to find the emotional instability and see that it is corrected. This is a safe method of solving the problem.

5. A psychiatrist is a very able adjunct in a great many cases of aphonia.

608 GUARANTY BUILDING.

XLIV

INTRALARYNGEAL OPERATION FOR CANCER OF THE VOCAL CORD

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It is with much fear and trepidation that I discuss the intralaryngeal operation for certain types of cancer of the larynx. Fear, because of the statement made years ago by one of our confreres that "intralaryngeal surgery for malignancies should be mentioned only to be condemned." Trepidation, because of my boldness in championing a condemned procedure. Experience over a period of many years has, in my opinion, definitely established a place for the intralaryngeal operation, provided the surgeon rigidly adheres to certain requirements in selecting the cases. Cancer of the larynx is still a major problem partly solved but not completely conquered. Eminent authorities have outlined the surgical methods generally accepted in the treatment of carcinoma of the larynx. The end results from these operations are truly astonishing, as emphasized by the late Dr. George Crile when he said that intrinsic carcinoma of the vocal cords offers a larger percentage of cures than carcinoma occurring in any other internal organ of the body provided an early diagnosis is made. Early diagnosis, then, is of paramount importance and universal acceptance of the dictum that "every case of persistent hoarseness should be considered potentially malignant until proved otherwise" would insure early diagnosis in most cases of cancer of the larynx.

The accepted surgical procedures for malignant lesions of the larynx are laryngofissure and laryngectomy. The selection of either procedure depends upon the location, type and extent of the lesion. Laryngofissure is an ingenious technical conception which yields excellent results when properly employed. I am thoroughly cognizant of its high merit and fully appreciative of its great value to laryngology. Laryngectomy is a heroic, life-saving operation which, unfortunately, must be performed all too frequently. While per-

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forming a laryngectomy, I am ever conscious of the fact that if the patient had received the proper attention at the proper time total removal of the larynx would not have been necessary.

My experience has proved that there are certain cases of carcinoma of the larynx in which a third type of operation can be performed with equally good, if not better, results than those obtained by laryngofissure, provided certain fundamental requirements are rigidly observed. This third type of operation is intralaryngeal dissection of early intrinsic carcinomas of the vocal cord. The technic is not original but was acquired during my association with the late Dr. R. C. Lynch[1] and it has now been used over a period of many years with such gratifying results that I believe it deserves serious consideration.

For the purpose of analyzing cases of intrinsic carcinoma of the larynx and in an effort to classify them into surgical groups more easily, it has been found convenient to divide them arbitrarily into three classes: (1) early cordal lesions, (2) cordal lesions and (3) cordal fixation or extension to the opposite vocal cord. Groups 2 and 3 represent those cases requiring laryngofissure and laryngectomy. There can be little or no disagreement in the selection of cases which fall into these two groups. This presentation is primarily concerned with the early cordal carcinomas which have been placed in Group 1.

Occasionally, one sees an early cordal carcinoma which appears so limited in extent that one wonders whether an operation as extensive as laryngofissure is necessary to eradicate so small a growth. Such a small growth, representing the earliest and most definitely limited malignant lesion encountered in the larynx, is confined to one vocal cord and may well be considered a surface lesion. These small surface growths are slow to infiltrate the surrounding tissue because of the usually low type of malignancy and the paucity of the lymphatic supply in this area. The growth may be only the size of a grain of rice, a pea or a dimpled match head, and it is usually situated near the junction of the anterior and middle thirds of the cord. However, it is not size alone which makes it amenable to intralaryngeal extirpation; in order to warrant such treatment it is absolutely necessary that healthy cordal tissue be visualized on all sides of the neoplasm. In a case of this kind, the histologic diagnosis may be made by rush biopsy and the amount of tissue to be removed will be governed by the principles on the extension of malignant tumors as set forth by New and Fletcher.[2] According to Clerf[3]

in 40 per cent of all patients with carcinoma of the larynx reporting to the laryngologist for the first examination, the lesion is so far advanced that little or no surgical treatment can be offered. When this is taken into consideration, it should be no surprise that few cases will be found to satisfy the requirements for intralaryngeal dissection. Briefly, these requirements are that the growth must be in the early stages of development, small in size, limited to one vocal cord and completely surrounded by normal cordal tissue. If there is any question about its being too extensive for intralaryngeal dissection, laryngofissure is advised. As one gains experience in intralaryngeal surgery, border line cases may be attempted, but they must be watched more carefully for the possibility of recurrence.

The prognosis of early cordal carcinoma is good because of the location of the growth, the type of malignancy and the lymphatic supply in this region. The growth is located on a limited portion of the surface of one vocal cord and is surrounded on all sides by healthy tissue. Because of the histologic structure of the vocal cord, 95 per cent of the growths are of the squamous cell variety; they are usually of low gradation and develop slowly. Since the lymphatic vessels of the true cords are small in size and number, there is no early metastasis.

The advantages of intralaryngeal extirpation are: (1) simplicity of operation, (2) no external incision, (3) preservation of continuity of the thyroid cartilage, (4) better postoperative phonation, (5) shorter hospitalization and (6) good prognosis.

Intralaryngeal extirpation for intrinsic cordal carcinoma is best accomplished by means of suspension laryngoscopy. This method affords excellent direct visualization of the vocal cords and permits the use of both hands for performing the operation. This approach and technic are largely responsible for simplifying intralaryngeal operations so that malignant lesions may be adequately treated. Once the larynx is exposed, the tumor is again carefully examined to check on its size and extent (Fig. 1). The vocal cord is retracted and the undersurface is examined. The tumor is then grasped with fixation forceps and traction is made toward the opposite side, thus placing the cord on medial tension while the tumor together with a wide margin on all sides of normal-appearing tissue is excised. Ambidexterity is a valuable asset in performing these operations. After the tumor has been removed, the edges of the wound are electrocoagulated and bleeding or oozing controlled (Fig. 2). Pentothal sodium is the anesthetic of choice in such cases because of the neces-

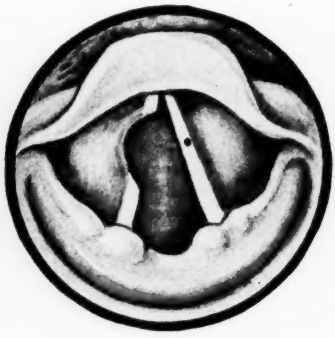

Fig. 1.—Early cordal carcinoma.

Fig. 2.—Appearance of larynx immediately following extirpation of cordal tumor.

sity of electrocoagulation. There is, as a rule, comparatively little postoperative reaction. Serious hemorrhage has never occurred and it has never been necessary to perform postoperative tracheotomy on any of the patients in my series. Vocal rest is observed and the patients are kept abed for several days.

During the healing process the filling defect in the anterior half of the vocal cord is gradually repaired. Occasionally, there develops granulation tissue, which, when persistent, may require removal and cauterization. Because of the small size of the tumor and the consequent limited portion of the cord removed, wound healing and repair rapidly occur (Fig. 3). In many cases a band of cicatricial tissue replaces the lost portion of the cord and occasionally the repair by nature closely resembles the original structure. With such satisfactory repair one can reasonably expect a voice far superior to that obtained in the average patient subjected to laryngofissure.

Patients who have had intralaryngeal extirpation are given the same scrutiny and careful postoperative observation accorded all patients with laryngeal neoplasms. They are not considered cured until five years have elapsed during which time it is imperative that the larynx be subjected to repeated examinations. Probably, the surgeon gets better cooperation from his patients and keeps his own thinking in the proper direction if, after treatment, patients with carcinoma of the larynx are considered "well without recurrence" rather than "cured." Only in this manner can an early recurrence

Fig. 3.—Appearance of cord when healed.

be discovered. Early recognition of recurrence followed by cauterization or the performance of laryngofissure might still preserve a crippled but precious and partially functioning organ. If the continuity of the larynx is destroyed by splitting the thyroid cartilage, recurrence of the neoplasm may be attended by serious developments.

I have operated on 39 patients with early intrinsic carcinoma of the vocal cords by the intralaryngeal route and electrocoagulated the base of the lesion following the operation. Nineteen of these patients have survived the five-year period after which they are considered "cured." Fifteen patients are living and well from one to five years after operation. Two patients have been operated on so recently that no result can be given now. Three patients have had recurrences; two required laryngofissure, and one later required laryngectomy. In this latter patient, 11 years after an intrinsic tumor of the left vocal cord had been successfully removed by intralaryngeal dissection, a tumor developed on the opposite vocal cord which, when first seen, showed fixation of the cricoarytenoid joint with involvement of the entire right side of the larynx.

With the extensive cancer program now being carried on throughout the nation it is reasonable to expect that more and more cases of early carcinoma of the larynx meeting the rigid requirements for intralaryngeal operation will be seen. Complete eradication of the malignant lesion with maximum preservation of organic function is the ideal and in early cordal cancers this can be achieved by intralaryngeal extirpation of the tumor.

OCHSNER CLINIC BUILDING.

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XLV

SOME CAUSES OF HOARSENESS IN CHILDREN

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One subject in laryngology that has not received sufficient attention is hoarseness, or other faulty voice conditions in pre-adolescents or young adolescents. We all seem to expect to hear the "cracked" voice in boys and have a tendency to laugh it off, and to assume that the lowering of the voice in girls is just part of the transition phase. It is not fair to these children to take this attitude and it is our duty to investigate these conditions, as many times there are associated remedial causes which should be eliminated. (Hoarseness, nasality or denasality, inability to pronounce words, and slurring of words in children has not been of major interest to laryngologists.) Appropriate treatment, plus intelligent instruction in voice improvement can frequently produce results which immeasurably improve the lot of these little sufferers. The general practitioner struggles along with such a case hoping the condition will be outgrown, and in this hope he is often aided and abetted by members of the child's family. Later, depending upon the sensitivity to ridicule, an inferiority complex of varying degree is developed, and the sufferer may thereby be handicapped both economically and socially. If pride in having a pleasant voice can be instilled in a child, instead of allowing fear and a sense of inferiority to dominate because of an unpleasant voice, then much can be done to aid our medical and surgical treatment. It is only with concerted action that the multitude of defective speech cases are discovered. This obtains in large centers where a Department of Speech Improvement is part of the public school system.

It is my privilege to be in close contact, in a voluntary capacity, with the Department of Speech Improvement in New York City. Over a period of many years, scores of school children were referred to my clinic at the Manhattan Eye, Ear, and Throat Hospital. It is interesting to note that about eight to ten per cent of the school children in the elementary schools, and about eight per cent in the

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high schools suffer from speech defects. Of these defects, hoarse voice is the most common. In general, more boys than girls are affected, and among stutterers the proportion may be eight to one. One survey of 50 stutterers showed that all but five were of superior intelligence. Unfortunately, all cases of hoarseness are not laryngologically examined, probably because of family neglect or lack of interest. About 50 per cent of the cases are seen by laryngologists.[1]

In examining the children at the Clinic a method, similar to the screening process recently used by the Armed Forces, was employed. The object was to try to find some underlying causes for the speech disorders and eliminate such pupils from group instruction while they are handicapped by remedial conditions. Not only was the time of the children and the instructor conserved, but the procedure also lessened the possibility of disrupting the morale of other class members who were not held back by similar handicaps. After some experience, the following syllabus was evolved to expedite the examinations by suggesting the ground to be covered.

Age: Instability of nervous system first manifesting itself at puberty.

Onset: Date and mode.

Corrective measures taken:

History of infectious diseases, trauma, psychic shock. A complete family history especially concerning neuroses.

Inciting causes: Congenital anomalies—malformations of larynx, cartilage, muscles, choanae, jaws, innervation, cleft palate, harelip, tongue-tie.

Environment:

Inflammations: Primary and secondary.

Mechanical obstructions:

Hearing difficulties: Unilateral or bilateral.

This was a comprehensive guide which could easily be expanded to individual needs to include laboratory tests, direct laryngoscopy, and bronchoscopy. At times, collaboration between the laryngologist, neurologist, ophthalmologist, and dentist was necessary to complete the diagnostic picture.

The ages ranged from 7 to 18 years, with an average of 14 years. This span of life is an important epoch, as development both physical

and mental should be normally progressive and at times, rapid. It is the period of greatest sensitivity, and psychic shocks and unfavorable impressions are carried over into adult life, acting as a cause of introspection. It is during these years that the infectious diseases of childhood are prevalent and may be the cause as, for example, paralysis following diphtheria, at times so mild it is not even considered. Chorea takes its toll.

Patients with congenital causes, such as cleft palate and harelip, were not referred to this Clinic. No case of an abnormally small larynx was seen, and extraordinarily, not one case of papilloma was seen. Only one case of tongue-tie was noted. Malocclusions required the attention of the dentist. Of the other congenital causes, the unusual case histories of four patients are of interest.

A girl, 14 years of age, was referred to the Clinic with the following history. Three years before she had suffered a febrile attack that kept her in bed for one week. She was not ill enough to have medical attention. A year later she complained of increasing hoarseness. An indirct laryngoscopy revealed a left abductor paralysis. She was referred to the Bronchoscopic Clinic where this diagnosis was confirmed, but no cause for the condition was discovered. All laboratory tests, including x-ray studies and fluoroscopy, were reported negative. She was referred to a neurological clinic in her own neighborhood (Queens General Hospital) and there, a diagnosis of possible postmyelitic paralysis was made. Later, she was admitted to the Neurological Institute for a complete study. The diagnosis was agenesis of the left vagus.

Two pitiful cases of congenital stenosis of the larynx were seen in siblings: a 16-year-old girl and a 14-year-old boy. They were referred to the Clinic for diagnosis and treatment. The girl's history was that at the age of seven weeks a tracheotomy was performed for a congenital web of the larynx. The boy had a similar condition and a similar operation was performed at the age of one week. In each case, it was impossible to examine them without the use of local anesthesia, and even then, only much deformed larynges without definite landmarks could be seen. The web in the girl's larynx occupied most of the anterior part. In the boy's larynx, only a small opening in the posterior part could be seen in the neighborhood of the arytenoids. The patients were referred to the Bronchoscopic Clinic, but not much more could be learned about their condition. Unfortunately, there was very little cooperation on the part of the patients even though a representative from their school tried very hard to induce them to allow us to proceed with treatments. The

last time I saw the girl was in the spring of 1945 when she was 18 years old. She was very much depressed. She had girl friends but no boy friends because of her "terrible" voice. Both patients were referred to Dr. Louis Clerf[2] at this time, as he had operated upon them, as noted above, and reported their case histories.

The fourth case history is that of a 15-year-old girl who came to the Clinic complaining of voice trouble: inability to phonate properly and a very hoarse voice. There was no demonstrable thyroid gland in the neck, but one was present on the base of the tongue. Because of its size, it acted mechanically on the larynx impeding its normal action and entirely obstructing the view into the larynx, so that only a very small portion of each arytenoid could be seen upon forced phonation. It was deemed advisable to try to shrink the mass in the region of the epiglottis by means of radium. She was referred to the Radium Clinic for treatment but, unfortunately, this made matters worse. Then electrocoagulation was tried, and three or four small areas in proximity to the epiglottis were coagulated. This procedure was carried out every two weeks for twelve treatments and at the same time thyroid extract was given by mouth. The gland in the region of the epiglottis was gradually reduced in size and the voice became clearer. The cords and the arytenoids became progressively easier to see, and the parts naturally involved in speech moved freely upon the slightest effort at phonation. The larynx became normal in color. The patient was under observation for two years, and during this time she grew taller with a proportionate increase in the size of the larynx. She was happy with the result when last seen, because her voice was clear and strong. Numerous other cases of goiter were observed but they were of the pretracheal type and also acted mechanically. In some cases, there seemed to be a sort of paresis of the epiglottis in which it quivered, but had no definite motion. In a few instances, the epiglottis would flap back against the posterior wall of the pharynx en masse or possibly, just the tip would do so during phonation. In all such cases medical treatment was supplemented by local laryngeal treatments as indicated.

One of the most important factors after obtaining a history—including age and evaluating the presence of an anomaly—is environment. Age and environment are naturally entwined as young children do not have much to say as to where they shall dwell. In other words, if the neighborhood is in proximity to an elevated railway and there is much trucking or other vehicular traffic in the streets, it is not only a noisy neighborhood but a dusty one as well. In any event, irrespective of the source of the noise and dust, a child able to

play in the street is not only going to be seen but is going to be heard, and that by yelling and screaming. Thus, more insult is added to an already overworked larynx. Home environment is also of importance. A sensitive child exposed to unhappy surroundings is at a great disadvantage in trying to become adjusted during the pre- or mutational stage. Most of the children seen at the Clinic were of the nervous or emotional type and often one or both parents were similarly affected.

The inflammatory reactions in the average case had the appearance of the so-called "rheumatic larynx," but pain was not a symptom in any case. In all instances the true cords, but most characteristically the arytenoids, were involved. They were red and moved very sluggishly, because of the inflammation and the trauma incident to excessive efforts to speak louder. Interarytenoid thickening was seen in only a few cases. Although most all of the cords approximated comparatively well, they could not hold their position except momentarily. "Head cold" was the most common complaint. Catarrhal and suppurative sinusitis were frequently associated conditions. The ever-present postnasal drip was in evidence and the intra- and extralaryngeal structures were bathed in the discharge. In the simple type of laryngitis, treatment was directed to the condition itself, but in laryngitis due to other causes, the larynx was treated concomittently. The desire of the child to talk above the laryngitis handicapped our efforts markedly. Not infrequently, children with a mild type of laryngitis stated they could get along pretty well until suddenly called upon to recite or answer questions. Due to emotion, plus the physical handicap of laryngitis, they could not phonate and were "scared stiff" as they expressed it because they knew they could not speak out loud under such circumstances. In all instances, vocal rest was insisted upon as far as possible, including the limiting or the elimination of recitations in classrooms, gymnasium work, and the enlisting of the cooperation of the members of the family at home.

Of the obstructive causes, tonsils and adenoids, with the presence of a deviated septum, naturally predominated. There was often a history of one or more tonsil and adenoid removals without beneficial results. In fact, this history became so common that, when given, instead of our examining the throat, a nasal examination was immediately made to ascertain if the real cause was not a nasal obstruction, possibly a deviated or markedly irregular nasal septum. The thyroid gland was always investigated, especially in girls, and in many instances it was found enlarged, causing pressure upon the larynx. Naturally, proper treatment was instituted.

Hardness of hearing, either unilateral or bilateral, is a potent factor in causing faulty speech. It is impossible to repeat correctly what one does not hear distinctly. The requirement to have regular and comprehensive hearing tests made in public schools is of inestimable help. A great responsibility lies with those of the teaching profession who are in contact with children having speech defects.

Attention is being brought to the fact that all changes in the voices of children are not to be classified as a part of the physiological changes either before or during puberty. The lasting ill-effects not only to the larynx but to the psyche of the person may be caused by failure to thoroughly investigate laryngologically all young persons suffering disagreeable changes in their voices. Much can be done to improve the speaking voice by cooperation between the laryngologist and the speech teacher.

An interesting aspect of this work has been the fact that so many children of various ages and nationalities could be examined by the indirect method of inspection. In other words, it can be done but it does take quite a little patience and, I might say, a little dexterity.

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XLVI

ENDAURAL APPROACH TO THE MASTOID FOR COMPLETE MASTOIDECTOMY, WITH REPORT OF 43 OPERATIONS

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Lempert[6] first described his technique in the subcortical or endaural approach to the mastoid in 1928, reporting 116 cases of mastoiditis. In 1938 he[7] reported the results of 1615 operations by a new and perfected technique of the endaural antauricular surgical approach to the temporal bone. He described in detail the incisions, opening, cleaning of the diseased temporal bone, and postoperative care. Since that time many reports have appeared showing results of operations performed by this approach. However, these reports have chiefly concerned the fenestration operation and radical and modified radical mastoidectomy. Few reports of the so-called simple mastoidectomy for acute mastoiditis have appeared.

Historically, the endaural approach is not new, for Thies[4] in 1933 states that Kessel reported its use as early as 1885. Kessel's incisions, however, were not the same as those of Lempert. The success of the operation is dependent entirely upon the technique of the incisions as described by Lempert, especially in the complete or simple endaural mastoidectomy.

Using this technique, Howarth and Bateman[2] in 1938 reported 20 cases of simple mastoidectomy. They concluded "We are convinced that this method of approach has many advantages over the posterior auricular route, and we have not had cause to regret its use in any case." It is instructive to note that their report includes a wide variety of pathological conditions.

"The endaural approach in the complete mastoidectomy," stated Henner[1] in 1939, "can be performed by an experienced operator so every type of mastoid pathology can be dealt with adequately. Further, this approach preserves the principle of wide open drainage and permits daily inspection of the mastoid cavity." He reported 16

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simple or complete mastoidectomies done by Lempert's endaural technique.

Using the same approach, Johnson and Zonderman[3] reported eight cases and summarized as follows: "The endaural approach for simple mastoidectomy appears to be entirely adequate. Certain features of the method have been presented to indicate a distinct advantage over the post-aural route, especially the greater freedom from deformity and the lesser postoperative discomfort. The patients prefer this method."

Expressing no regret in using Lempert's endaural approach, Kettel of Denmark,[5] in 1941 reported 18 simple mastoidectomies. He stated, "The Lempert endaural approach to the temporal bone offers the great advantage that the surgeon is enabled at once to resect any cell system, regardless of extent, just as through the post-aural incision, and at the same time to have a direct view of and access to the tympanic cavity. Moreover, this operation gives an extraordinarily fine result cosmetically."

Experience of an older and a younger man was related in 1942 by Woodruff and Henner,[8] who reported on 76 endaural operations, 29 of which were for acute mastoiditis. Henner, the younger man, states "The endaural route is the method of choice in complete mastoidectomy for the following reasons: (1) since acquiring experience in the method there has been no difficulty in adequately handling any type of pathologic condition in any area of the mastoid; (2) the after care is simplified; (3) the minimal trauma to normal tissues and the maintenance of wide open drainage appear to lessen postoperative complications." Woodruff, the older man, feels that "the question of the choice of method roughly boils down to this: the complete mastoid operation is more difficult to perform by the endaural than by the post-auricular route. On the other hand, the period of convalescence following the endaural operation is usually easier for the patient and simpler for the surgeon than that following a post-auricular operation." After careful consideration based on his own experience he concluded: "(1) Proficiency in the endaural complete mastoidectomy is gained only after thorough study of the method, followed by carefully performing at least 10 to 12 such operations on the cadaver. (2) A surgeon thoroughly proficient in the endaural method can adequately carry out all steps of the complete mastoid operation, and is then justified in employing the method, thereby securing the advantages of a more favorable postoperative course. (3) A surgeon who has not acquired this proficiency should not attempt to use the endaural route for com-

plete mastoidectomy. The operation in his hands is not a safe procedure. He may easily do incomplete work or injure important structures."

The purpose of this paper is to report experience with the endaural approach in operations for acute mastoiditis only. I have now performed 43 operations for acute mastoiditis by the endaural route. The patients ranged in age from 2 to 74 years. The first 20 patients were studied by x-ray examination not only before but also after operation. The examinations were done to make sure that the mastoid cells were exenterated as completely by the endaural as by the postauricular technique. All anatomical varieties of mastoid were encountered in this study. Using Lempert's technique I found no difficulty in gaining access to any pathologic condition of the temporal bone. I wish to emphasize, however, the necessity for correct incisions and thorough mobilization of the ear.

I have been asked about the time taken for the operation as compared with the postauricular approach. To my mind, the time element neither condemns nor praises any operation. However, I have found that after one has become skilled in the endaural incisions and operative technique, the time of the two operations is about the same. It takes much longer to make the incisions in the endaural approach, but that is compensated for by the fact that no time is taken for closing. Furthermore, time consumed in changing dressings is reduced to a minimum, for only two or three changes are necessary in the average case. After the third or fourth day no more dressings are necessary; only a small piece of cotton is placed in the ear to be changed as required.

No instance of lateral sinus thrombosis was encountered, but in the 74-year-old man, who had a very wide mastoid, I found a large perisinuous abscess covering practically the entire mastoid portion of the sinus. The diseased bone was cleared from the entire sinus and, surely, if that could be done, the sinus could have been opened and cleared of its thrombus.

Edema of the skin and consequent difficulty in exposure of the tip of the mastoid process were encountered by both Kettel and Henner in patients in whom the disease was in an early stage. This has not been a problem in my experience, although some types of ear canal make the approach somewhat more complicated. A small ear canal makes the exposure harder and the second incision may have to be extended over the temporal muscle. However, lengthening of

the incision apparently makes little difference in the appearance of the scar.

One patient with Bezold's abscess was seen in my series. The abscess was drained through the endaural opening, thus eliminating the customary external incision.

Zygomatic cells were found in several patients and were readily reached. Complete exposure is obtained without extension of the incisions or trauma to the temporal muscle, facts which emphasize that the endaural route is an ideal approach to the zygomatic area.

Several subperiosteal abscesses were encountered in this series, and all patients with this condition recovered uneventfully.

In discussions of Lempert's endaural approach, the question of cartilaginous infections often arises. This complication has not been observed in my series. In his report of 1938 Lempert stresses the point that this approach is extracartilaginous. Therefore, let me again emphasize the importance of the correct incisions. It must be remembered that the cartilage is always cut in the posterior approach for a radical operation, and in my experience infection of the cartilage has not occurred.

All my patients have had much less postoperative pain or discomfort. Seldom are opiates necessary following the operation. In fact, the younger patients sit up in bed, read or play as soon as they have recovered from the anesthetic. They are allowed bathroom privileges on the second day and are permitted to be up and around as soon as the temperature returns to normal. Normal temperature returns much earlier in patients operated upon by the endaural approach, provided theirs is an uncomplicated case, than in those operated upon by the posterior route. In my opinion the more satisfactory decline of fever is due to adequate drainage without foreign bodies, such as stitches and drainage tubes, in the septic wound.

The patients in this series were little more disturbed by the operation than by the previous myringotomy. The lack of postoperative pain is due, in my opinion, to several factors: (1) no stitches are placed in a septic wound to aggravate inflammation; (2) no drain is used in the septic wound because the opening is adequate for complete drainage; and (3) because the upper fibers of the sternomastoid muscle are not cut and only the periosteum is separated from the tip, no muscle pain results. Two patients who had been operated upon previously by the posterior route for simple mastoiditis stated that there was much less pain following the end-

aural operation. Each regained strength and resumed work earlier than after the previous operation.

Because the patients are ambulatory much earlier than when the posterior approach is used, hospitalization is shortened to an average of five to seven days, and return to school and work is expedited.

A distinct advantage of the endaural route is the ability to observe the entire deep interior of the wound at will. This is accomplished by placing a large sized ear speculum in the wound.

Deep, depressed scars and even fistulas are not infrequent following posterior route operations. These sequelae cannot occur after the endaural operation. So it seems to me that in this age, when so much cosmetic surgery is being advocated, this is an important point. To be sure, in some cases there is a small scar between the tragus and the helix, but I have not seen any but what had practically disappeared in six months' time.

Atresia of the canal is sometimes feared as a possible complication of Lempert's operation. If one follows his technique exactly this complication will not occur. In one of my patients on whom a double mastoidectomy was performed, I made an incorrect incision. In that ear there was a slight narrowing of the canal. I have seen a number of collapsed or narrowed canals following posterior route mastoid operations done by well-qualified surgeons.

Two patients of the series came with postoperative fistulas, following previous posterior mastoidectomy. These fistulas were repaired easily in the endaural operation. The resulting scars were cosmetically more satisfactory than any I have obtained by any other method.

SUMMARY

This small series of cases has shown that:

1. The endaural route is excellent in complete or simple mastoidectomy, because pathological conditions of the temporal bone can be reached easily by this approach.

2. There is less postoperative pain and easier convalescence.

3. The head dressing can be discarded much sooner, making the patient more comfortable.

4. The hospital stay is shortened, reducing expense and time loss.

5. The endaural route avoids the occasional depressed and unsightly scars and postauricular fistulas.

CONCLUSION

If one follows closely all the steps as described by Lempert, then the endaural approach to the mastoid is ideal as far as I have been able to discover in my series of cases. I believe that anyone attempting endaural surgery should have mastered the technique on cadavers. The three incisions should be made with great care and complete mobilization of the ear should be accomplished; then and only then is it possible to judge the merits of this approach.

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XLVII

ASSOCIATED PARALYSES OF THE LARYNX

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The paralyses of the larynx may be divided into three groups. The largest is that of paralysis of the recurrent nerve; in this type there are no other neurological signs. The second is paralysis of the vocal cord; this paralysis is merely one of many symptoms occurring in some of the systemic nervous diseases. Multiple sclerosis, pseudobulbar paralysis, syringobulbia, locomotor ataxia, the bulbar form of progressive muscular atrophy, apoplexy and the bulbar type of acute poliomyelitis may present at some stage a paralysis of the vocal cord as one of many other findings. The smallest group is the associated paralyses of the larynx; in this type there are neurological symptoms from various combinations of lesions in the last four cranial nerves. The associated paralyses of the larynx may be classified as the laryngeal syndrome of the medulla and the laryngeal syndrome of the jugular foramen. There are several syndromes of the medulla in which the motor nerves to the larynx are not involved. This classification includes the eponymic syndromes from lesions in the medulla oblongata as well as those at or near the jugular foramen.

Jackson[1] in 1864 reported a case showing homolateral paralyses of half of the soft palate, half of the tongue with atrophy, of the vocal cord and of the sternomastoid and trapezius muscles. The laryngoscopic examination was made by Morell Mackenzie who described a complete paralysis of the vocal cord. Mackenzie[2] in 1880 reported a patient who had been seen with Jackson in 1868, whose case now would be diagnosed as Tapia's syndrome. Mackenzie also differentiated between lesions in the medulla oblongata and those at or near the jugular foramen. In 1886, Jackson[3] reported a series of cases one of which would now be called the syndrome of the posterior inferior cerebellar artery. It seems clear that for some 30 years the English laryngologists recognized the associated paralyses of the larynx, understood the etiology, located the lesion and regarded cer-

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tain groups of symptoms not as separate entities but merely as variations of essentially the same condition. It is regrettable that this view did not persist. Then the Germans and later the French individualized these groups of symptoms almost into clinical entities as eponymic syndromes. Avellis[4] reported the syndrome which bears his name in 1891, Schmidt[5] in 1892, Tapia[6] in 1906, Vernet[7] in 1915, Collet[8] in 1915 and Villaret[9] in 1917. Since that time, the American laryngologists have tacitly refused to add any more eponymic syndromes to this collection and so add further to the confusion already prevailing.

The incidence of the associated paralyses of the larynx is rare. The consolidated reports on paralyses of the larynx by New and Childrey,[10] Suehs[11] and Work[12] total 734 cases, of which 30 were the associated paralyses, or four per cent of the total. The laryngeal syndrome of the medulla occurred in 21 of these 30 cases, and the syndrome of the jugular foramen in 9. However, the relative incidence of the associated paralyses and the syndromes of the medulla and jugular foramen varies considerably in the respective reports. Thus it is apparent that if a clear picture is to be obtained of the subject as a whole, reference must be made not to personal experience but to the literature, unsatisfactory though this is.

The etiological factors in the laryngeal syndrome of the medulla are infections and vascular disease. The most frequent infection causing this syndrome is the bulbar type of acute poliomyelitis. Syphilis is next in frequency and is regularly manifested as endarteritis of the arteries supplying the medulla, though a gumma is sometimes present. Tuberculosis has occurred as a tuberculoma of the nucleus ambiguus. Thrombosis of the arteries supplying the medulla is statistically less frequent than lues, but such was not the case in my limited experience. Vasoconstriction of the radicular arteries to the motor nuclei must be added to thrombosis as a vascular condition. One of my patients had a sudden onset with paralysis of half of the soft palate, half of the tongue, an abductor paralysis of the vocal cord and complete loss of voice. When examined seven hours later, the palate, tongue, vocal cord and voice were normal. Vasoconstriction of the medullary arteries may be more frequent than is indicated by reports in the literature. Hematobulbia occurs in extensive lesions and seems to be included usually under the diagnosis of thrombosis.

The causes of the laryngeal syndrome of the jugular foramen are quite diverse. The chief cause is a neoplasm usually from the

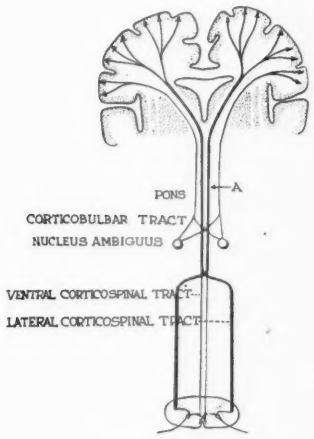

Fig. 1.—Schema of bilateral cortical innervation of each nucleus ambiguus.

nasopharynx but occasionally from the tympanum. The next in order of frequency is thrombophlebitis of the jugular bulb. Other factors are abscess in the parapharyngeal space, tuberculosis of the cervical glands, fracture of the base of the skull, and gunshot and stab wounds.

The symptomatology of the associated paralyses of the larynx is dependent upon the location and extent of the lesion in the medulla oblongata, or at or near the jugular foramen. The medulla oblongata containing the nuclei of the last four cranial nerves, decussation of the motor and sensory tracts and the paths of other motor and sensory tracts is the switchboard and relay station of the nervous system. The extension of a lesion in this area may occasion many combinations of neural and laryngeal symptoms. The nucleus ambiguus is, of course, invariably affected in the laryngeal syndrome of the medulla. For this nucleus contains, as it descends through the medulla, the origins of the motor fibers of the ninth, tenth and eleventh cranial nerves in the order named. A lesion extending to successive levels of this nucleus causes a combination of lower motor neuron or flaccid paralysis of the glossopharyngeal, vagus and accessory nerves. To illustrate, a lesion in the lower part of the nucleus ambiguus produces a unilateral flaccid paralysis of the vocal cord;

if it extends upward, half of the soft palate is paralyzed. But if it extends downward, torticollis or wry-neck is also present.

The upper motor neuron of the nucleus ambiguus is the corticobulbar tract which arises in the inferior precentral area of the cortex. As a part of the corticobulbar tract is another tract, designated the aberrant pyramidal by Dejerine in 1914, which descends through the medulla in or near the medial lemniscus. The latter tracts at successive levels usually have a bilateral or bridle decussation to each nucleus ambiguus as shown in Fig. 1. Thus the vocal cords usually have a bilateral cortical innervation as first demonstrated by Krause in 1884 and since confirmed by many anatomists. A lesion of one corticobulbar tract, marked "A" in the schema, usually does not cause a unilateral paralysis of the vocal cord; but if sufficiently large to involve both corticobulbar tracts may cause a bilateral spastic paralysis of the vocal cords. This supranuclear innervation of the nucleus ambiguus is not unique, for all other pontinebulbar nuclei of cranial nerves to muscles which move bilaterally to perform their functions likewise have a bridle decussation. The corticobulbar and aberrant pyramidal tracts are subject to considerable individual variations. While the bridle decussation to the nuclei ambigui usually occurs, it is by no means invariable. Furstenberg[13] has pointed out that a unilateral supranuclear lesion may cause a unilateral spastic paralysis of the vocal cord. A unilateral lesion of the vocal cord is presumptive evidence but hardly positive proof that a supranuclear lesion does not exist. His clinical experience thus confirms the anatomical dissections.

The relation of the nucleus ambiguus to the tracts and other nuclei accounts for the wide diversity of symptoms encountered in the syndrome of the medulla. The schematic outline of the location of other nuclei and tracts usually involved in this syndrome is shown in Figure 2. The blood supply of the medulla is derived from the vertebral artery through its branches, the anterior spinal, the posterior spinal and the posterior inferior cerebellar arteries, and to a lesser extent from the basilar. The anterior spinal artery through its central branches supplies the pryamid, the medial lemniscus, and the inferior olive, and its radicular branches accompany the fibers of the cranial nerves to their nuclei. The posterior spinal artery supplies the choroidal plexus and sends branches to the nuclei near the floor of the fourth ventricle. The branches of the posterior inferior cerebellar artery pass through the lateral wall to supply the superficial and deeper tracts. The nucleus ambiguus through the long and slender radicular arteries is more exposed to vascular disorders and

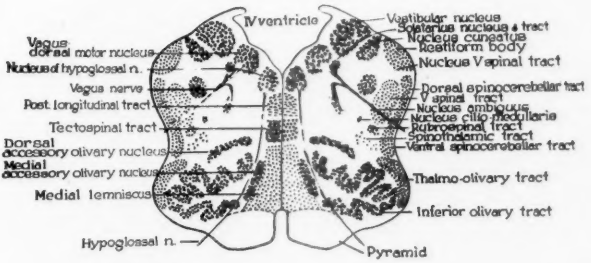

Fig. 2.—Schematic location of nuclei and tracts in the medulla oblongata. (Modified after Ransom)

so is more frequently affected. The nuclei on the floor of the fourth ventricle have a larger arterial supply than any other area and so are less often involved. The anatomy of the arterial supply is important clinically in locating the site and extent of thrombosis and hematobulbia.

LARYNGEAL SYNDROME OF THE MEDULLA

The one constant symptom in the laryngeal syndrome of the medulla is hoarseness. The onset is sudden. Additional symptoms appear as the lesion may extend from the nucleus ambiguus to other nuclei and tracts. The voice may have a nasal tone, articulation may be poor or aphonia may be present. The paralysis is flaccid. An abductor paralysis is present and the affected vocal cord may be fixed in the intermediate or even in the mediate position. The arytenoid may be tipped forward, lying slightly below its fellow. On the other hand, the affected cord in the intermediate position may on phonation approximate the midline and lie slightly below its fellow. The innervation of the interarytenoid muscle by a branch of the recurrent nerve of the opposite side causes the adduction. The laryngoscopic appearance is a paralysis of the superior and inferior laryngeal nerves. Anesthesia of the affected vocal cord is variable.

The degree of dysphagia varies from slight inconveniences to regurgitation through the nose. Unilateral paralysis of the soft palate, tongue and pharyngeal constrictors accounts for the difficulty in swallowing, but not for the regurgitation in some and its absence in others. Imperatori[14] may have given the lead to the explanation, for he seems to have been the first to report a spasm of the cricopharyn-

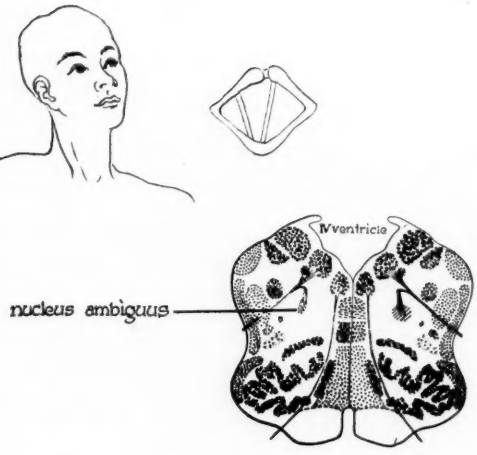

Fig. 3.—Schema of Schmidt's Syndrome.

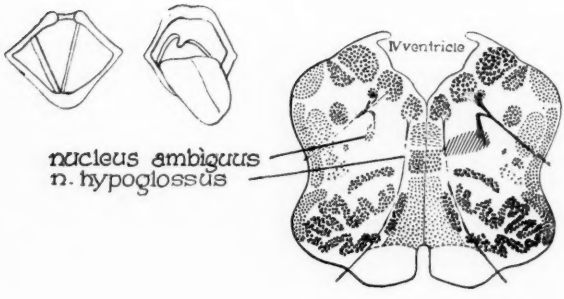

Fig. 4.—Schema of Tapia's Syndrome.

geus muscle. This suggests a reflex action analogous to a hyperactive knee-jerk.

Eponymic Syndromes. The description of the eponymic syndromes[15] begins with the one in which the lesion affects only a part of the nucleus ambiguus. The extension of the lesion is traced step by step until a large part of the medulla is involved. To the extent of being repetitious, a practical demonstration is thus afforded of the difficulty in associating a syndrome with the correct proper name. A nomenclature that is easy to remember and is associated with the area of the lesion is desirable.

Schmidt's syndrome is hoarseness, torticollis and drooping shoulder. Homolateral paralyses of the vocal cord and the sternomastoid and trapezius muscles are present. The lesion is confined to a part of the nucleus ambiguus and involves the vagus and accessory nerves. A variation shows paralysis of half of the soft palate with involvement of all of the nucleus ambiguus.

Tapia's syndrome is hoarseness and dysphagia. On phonation, the unaffected half of the soft palate and uvula is elevated, while the paralyzed part is drawn over as a curtain. The tongue on protrusion deviates toward the affected side and later atrophies. Homolateral paralyses of the soft palate, tongue, pharyngeal constrictors and larynx are present. The lesion involves the nucleus ambiguus and the nucleus hypoglossus.

Jackson's syndrome is hoarseness, difficulty in swallowing, deviation of the tongue, torticollis and drooping shoulder. Homolateral paralyses of the soft palate, tongue (with atrophy later) pharyngeal constrictors, larynx and sternomastoid and trapezius muscles are present. The lesion affects the nucleus ambiguus and the nucleus hypoglossus and involves the vagus and the accessory nerves. Loss of taste on the posterior third of the affected side of the tongue indicates an involvement of the glossopharyngeal nerve, but testing for taste is uncertain. One of my patients, E. B. C., had the classical symptoms associated with this group.

Avellis' syndrome is hoarseness or aphonia; dysphagia may or may not be present. The patient notices a loss of pain and temperature sensations on half of the body below the face. Homolateral paralyses of the soft palate and larynx are present with a contralateral loss of pain and temperature sensibility. The lesion affects the nucleus ambiguus and the spinothalamic tract. The syndrome has several variations, in one of which there are also homolateral enoph-

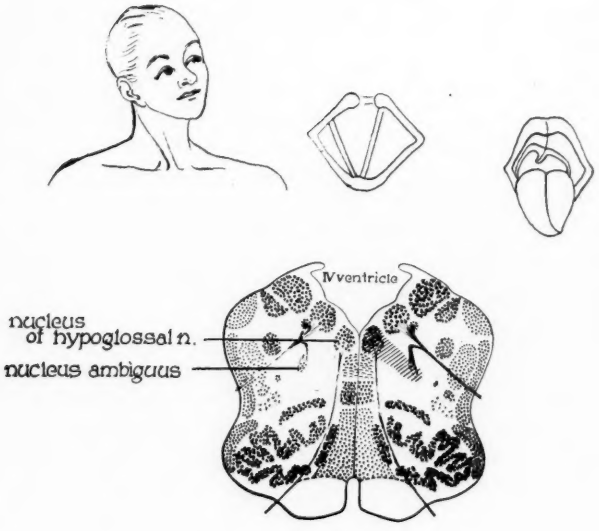

Fig. 5.—Schema of Jackson's Syndrome.

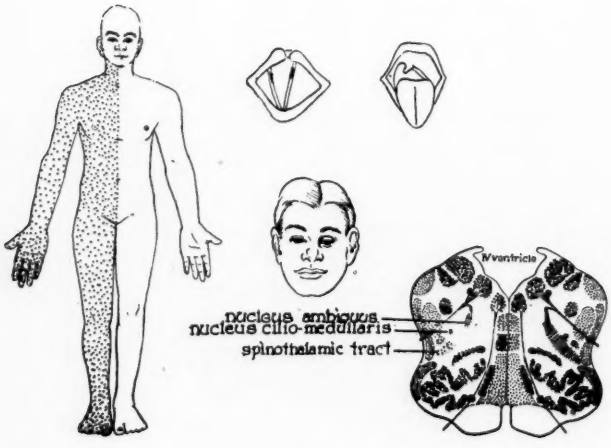

Fig. 6.—Schema of Avellis' Syndrome.

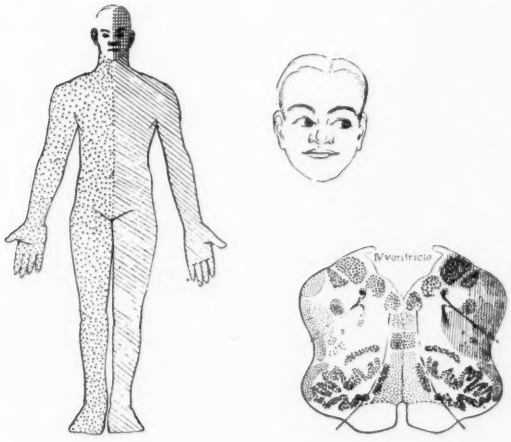

Fig. 7.—Schema of the syndrome of the posterior inferior cerebellar artery.

thalmos, pseudo-ptosis and miosis—a pseudo-Horner's syndrome—from involvement of the nucleus ciliomedullaris. One of my patients, R. A. W., presented this variation of the syndrome. In another variation there is also vertigo and spontaneous nystagmus. Burger[9] emphasizes the importance of a laryngeal examination in all cases of vertigo and nystagmus in order to differentiate brain tumor from the laryngeal syndrome of the medulla.

Wallenberg's syndrome is now regularly referred to as the syndrome of the posterior inferior cerebellar artery. The symptoms are hoarseness or aphonia, dysphagia with regurgitation, and asynergia, lateropulsion, ataxia, vertigo, nystagmus and conjugate deviation to the affected side. Homolateral paralyses of the soft palate and larynx are present from a lesion in the nucleus ambiguus. Homolateral vertigo, falling, nystagmus or conjugate deviation occur from a lesion affecting the descending vestibular tract or the nucleus. Homolateral asynergia and lateropulsion are from a lesion in the dorsal and ventral spinocerebellar tract, and ataxia from the rubrospinal tract. Homolateral enophthalmos, miosis and pseudoptosis occur from a lesion in the nucleus ciliomedullaris. Homolateral anesthesia of the face occurs from a lesion in the descending tract of the trigeminal nerve. Contralateral loss of pain-temperature sensibility is from a lesion in the spinothalamic tract. The symptoms

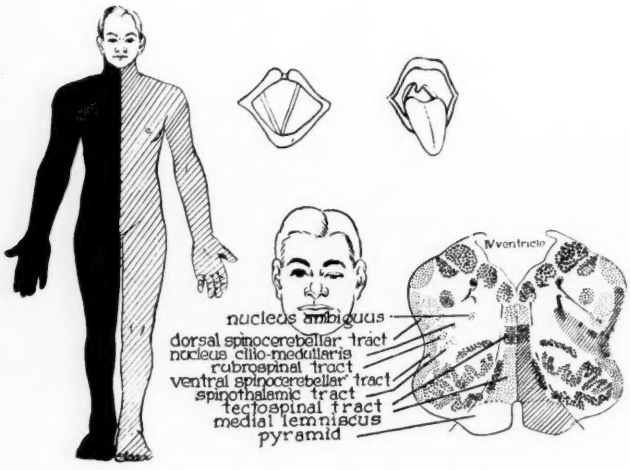

Fig. 8.—Schema of Cestan-Chenais' Syndrome.

from a thrombosis of the anterior spinal artery added to this syndrome are the same as those from a thrombosis of the vertebral artery.

Cestan-Chenais' syndrome is the result of multiple lesions in the medulla from degenerative processes persisting after a thrombosis of the vertebral artery. Homolateral paralysis of the soft palate and larynx is caused by a lesion in the nucleus ambiguus. Homolateral enophthalmos, miosis and pseudo-ptosis are from a lesion in the nucleus ciliomedullaris. Homolateral asyneria and lateropulsion are from the dorsal and ventral spinocerebellar tract, and ataxia from the rubrospinal tract. Contralateral loss of pain-temperature sense is from a lesion in the spinothalamic tract. Contralateral loss of discriminative, proprioceptive and tactile sensibility is from the involvement of the medial lemniscus. Contralateral hemiplegia is from a lesion in the pyramid.

LARYNGEAL SYNDROME OF THE JUGULAR FORAMEN

The laryngeal syndrome of the jugular foramen is the associated paralysis from a lesion at or near the jugular foramen. The eponymic syndromes tabulate only the last four cranial nerves; but the optic and the fifth to the eleventh cranial nerves were involved in one of

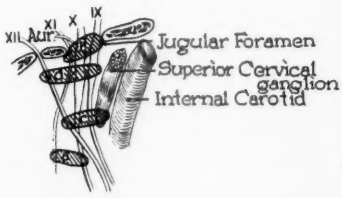

Fig. 9.—Schema of syndrome of the jugular foramen.

my cases, and other reports have implicated all of the cranial nerves except the olfactory. The onset may be sudden or slow. The sensory functions of the nerves are regularly impaired along with the motor. The sense of taste on the posterior third of the tongue on the affected side is lost, and in one of my patients the ageusia was complete. The pulse and respiratory rates are affected if the onset is sudden. I have had three patients with this syndrome: one, M. S. H., with thrombophlebitis of the internal jugular vein, who recovered; another, R. H. W., with carcinoma of the nasopharynx, who died; another C. H., with chordoma of the nasopharynx, who is living but unimproved. M. S. H. with thrombophlebitis had Vernet's syndrome but the other two cases do not conform to any of the eponymic syndromes.

The larynx was observed in each of these from the inception of the neural symptoms. M. S. H. with thrombophlebitis had an abductor paralysis with the vocal cord in the intermediate position and on phonation it approximated the midline. The larynx became normal with recovery from the thrombophlebitis. R. H. W. with carcinoma had an abductor paralysis with the vocal cord fixed in the intermediate position. C. H. with chordoma has an abductor paralysis with the vocal cord in the mediate position.

The schema (Fig. 9) illustrates the relations of the last four cranial nerves with each other and the superior cervical sympathetic ganglion. The sympathetic ganglion is shown to be affected only in Villaret's syndrome, but it may be in the others. The graphic identification of a syndrome is to show the nerves involved and not the location of the lesion. The lesions may be at or near the jugular foramen or in the parapharyngeal space.

Eponymic Syndromes. Garel-Gignoux's syndrome is hoarseness, torticollis and drooping of the shoulder. The unilateral paralysis of

the vocal cord is from damage to the recurrent nerve. The unilateral paralysis of the sternomastoid and trapezius muscles is from the accessory nerve. The syndrome is marked "a" on the schema.

Vernet's syndrome is hoarseness, dysphagia, torticollis and drooping shoulder. Paralysis of the glossopharyngeal nerve causes ageusia on the posterior third of the tongue and anesthesia about the base of the tonsil. Unilateral paralysis of the soft palate, the pharyngeal constrictors and the larynx follows paresis of the vagus. Paralysis of the sternomastoid and trapezius muscles follows involvement of the accessory nerve. The syndrome is marked "b" on the schema.

Villaret's syndrome is hoarseness, dysphagia, ageusia, deviation of the tongue on protrusion and later atrophy. Horner's syndrome is present following involvement of the sympathetic ganglion. The syndrome is marked "c" on the schema.

The syndrome of Collet-Sicard is a hemiplegia of the last four cranial nerves. It is marked "d" on the schema.

CONCLUSION

Who can remember the correct proper name to apply to a particular syndrome? The eponymic syndromes no longer facilitate the diagnosis of this condition, but act as an unnecessary handicap. As demonstrated here, their continued use is needlessly tedious and repetitious. The recognition of the laryngeal symptoms is unmistakable, but fitting them with the correct proper name is a puzzle not a diagnosis. The Babinski-Nageotte syndrome plus an involvement of the nucleus ambiguus is the same as that of Cestan-Chenais. The nomenclature should be revised, and instead of the eponyms there should be employed terms easy to remember with an application to the involved area.

The term increasing applied to this condition for the past 30 years has been associated paralyses of the larynx. This is awkward, may be confused with the laryngeal symptoms of systemic nervous diseases and particularly does not conform to the usual form of medical terminology. It is suggested that the name to be applied to these paralyses in the future could well be synoplegia laryngis (*syn* a prefix indicating united or associated and *plegia* a stroke or paralysis). The laryngologist would have a term equally applicable to all syndromes whether the lesion was in the medulla or at or near the jugular foramen. The eponymic syndromes could then be en-

shrined in medical history in grateful appreciation to the skillful clinicians who first described them.

1304 WALKER AVENUE.

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XLVIII

AIDS IN RHINOPLASTIC PROCEDURES

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The basic principles of rhinoplasty were formulated many years ago by Jacques Joseph of Berlin. The nasal plastic procedure of today differs from the technique of Joseph only in certain details. These details, however small, are important when they lead to a better end result.

Planning the Rhinoplasty. In planning the rhinoplasty a variety of procedures for analysis of the deformity have been advocated. Joseph devised a profilometer and also marked off profile angles on photographs. Modifications of these have been devised and published.

It is difficult to measure a subjective abstraction such as beauty in angles or degrees. There are too many variables in facial physiognomy and general body build which enter into the composite picture.

A method I have employed for a number of years has been of value in planning a rhinoplastic procedure. It consists simply of drawing a sketch of the new profile on the back of the patient's photograph. Standard 4x6 inch photographs are taken of both profiles, the front view and another taken from below to show the nostrils. The photographic printing paper should not be too heavy, so that its transparency will not be interfered with when the picture is held up to the light. A pencil sketch is then made as the photograph is held on an x-ray shadow-box. If this is not available a sketch may be made by holding the photograph up to a light or on a window. The original profile is lightly traced on to the back of the photograph and then a new profile line is drawn over this tracing. After several such sketches one can determine the profile best suited to the face under consideration. The new profile decided upon is then pencilled in a heavier line than the original sketch of the deformity, (Fig. 1). By again holding the photograph to the

From the University of Illinois, College of Medicine, Department of Otology, Rhinology and Laryngology.

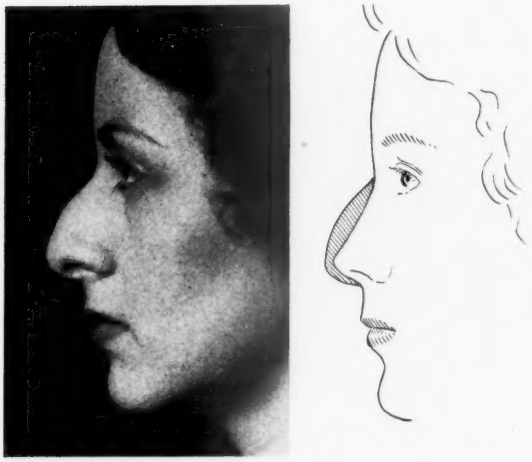

Fig. 1.—(A) Preoperative profile view. (B) Desired result as it is sketched on the reverse side of photograph (A) while it is held on an x-ray shadow-box or up to a light. From this procedure, one may readily judge the amount of tissue to be removed; also, the patient may be given a fairly accurate idea of what to expect as a result of the operation.

light one can readily judge the amount of tissue which must be removed or added, as the case requires.

The advantages of this method are:

1. It is simple.

2. Any number of sketches may be studied.

3. The sketch placed on an x-ray shadow-box in the operating room serves as a guide during the operation.

4. The patient may be given a fairly accurate idea of the result which can be expected.

A further analysis, aside from the esthetic, can be made from this simple study. On the second profile view, the nasal bone, the upper and lower lateral cartilages, and the approximate length of the septum can be sketched into the drawing, and the role played by each structure in the deformity may be fairly accurately evaluated and certain questions postulated and answered. Is the deformity

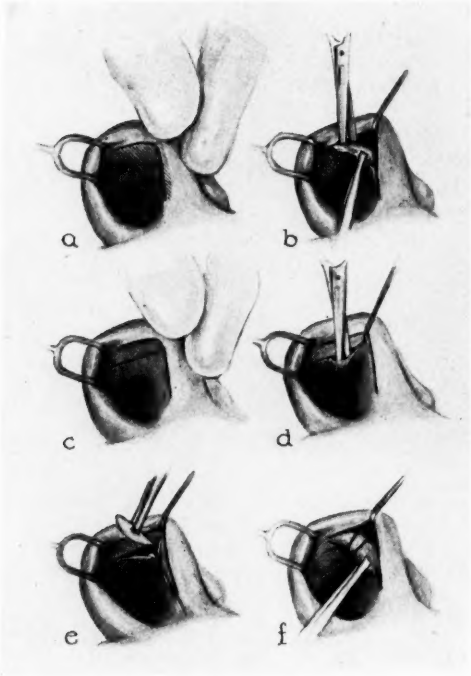

Fig. 2.—Correction of enlarged alar cartilages.

(a) Incision along anterior border of medial crus and border of lateral crus.

(b) Cartilage dissected from overlying skin.

(c) Lateral crus split transversely, separating it into an upper and lower field.

(d) Skin on under surface of upper segment dissected away from the cartilage.

(e) Removal of upper segment cartilage free of skin.

(f) Lower (rim) segment pulled outward and wedge of cartilage with skin attached removed from the angle between medial and lateral crura

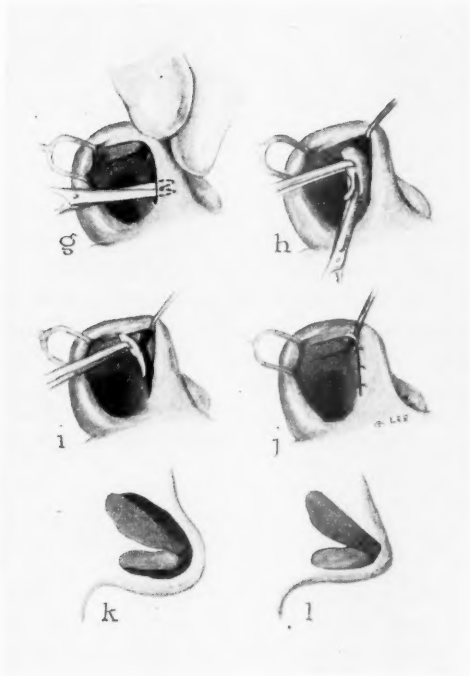

Fig. 2.—Correction of enlarged alar cartilages.

(g) Medial crus freed in its entire length.

(h) Cartilage, with skin attached, cut from the medial crus to aid in shortening and improving contour of the nasal tip.

(i) Section of skin and cartilage removed.

(j) Three fine black silk sutures uniting columellar skin to skin of remaining medial crus.

(k) View of alar cartilage in a large tip. Darker areas indicate amount of cartilage to be removed.

(l) View of reconstructed alar cartilage.

mainly bony or cartilaginous? How much of each? What type of alar cartilages are present? How much of the septum is it necessary to remove in the shortening of the nose? Is the septum hanging? The relation of the type of chin, the length of the upper lip and the type of forehead, important factors in planning the rhinoplasty, may also be studied.

In the front view, also on the reverse side of the picture, a few guide lines may be sketched as to the width of the nose and a straight line may be drawn down the center to determine any deviation of the bony or cartilaginous dorsum. In the nostril view, a sketch can be made to determine the amount of narrowing needed by drawing in the alar cartilages; also from this view, it can be determined whether the base of the nostril requires narrowing. Too much reliance cannot be placed on the latter two sketches, therefore, I prefer to draw guide lines directly on the skin of the nasal tip at the time of the operative procedure after the method described by Aufricht.[1]

Although this does not constitute an exact analysis, a fairly good idea of what is to be done is obtained before undertaking a rhinoplastic operation. Esthetic sense and judgment in any rhinoplastic undertaking is paramount.

The Alar Cartilages. Shaping of the nasal tip is one of the most important procedures in obtaining a natural, "non-surgical" appearing nose. Not infrequently after a proper removal of the hump and fracture of the nasal bones, the result is unsatisfactory because of inadequate or faulty modeling of the lower lateral cartilages. Considerable judgment and experience in correctly evaluating the proper shaping of the lower lateral cartilages is essential because no two cases are alike. A procedure which I have employed to advantage is a modification of the technique of Joseph[2] and Aufricht.[1] Essentially it embodies the principle of splitting the lower lateral cartilages into two fields, an upper and lower, the removal of a wedge at the angle of the two crura and of a segment of the anterior border of the medial crus.

The procedure which I have used when there is a large tip and a long nose, and the hump has already been removed is as follows:

1. The nasal tip is marked with methylene blue, delineating the alar cartilage into an upper and a lower field. The upper field includes almost half the cartilage, the amount to be removed depending on the height of the alar dome. The border of the lower field is marked at the angle of the cartilage (i.e., the junction of medial

and lateral crura), and the approximate amount to be removed to lessen the width is marked.

2. An incision is made, starting at the lowest point of the anterior border of the medial crus and following the border of the medial crus along its entire height to the junction of the lateral crus (Fig. 2a). The incision then follows the lateral crus for about three quarters of its length laterally.

3. The cartilage is then dissected free from the overlying skin by means of a curved Joseph scissors (Fig. 2b).

4. The cartilage is projected outward by a Joseph alar double hook and the lateral crus is split transversely (Fig. 2c). This incision should correspond to the first outside marking, and separates the cartilage into an upper and a lower field. The skin on the under surface of the upper segment is separated from the cartilage and this segment of cartilage is removed without sacrificing the skin (Fig. 2d and e).

5. The lower (rim) segment is then pulled from its bed and split at the angle between the medial and lateral crura. The necessary amount required for narrowing is removed from the lateral crus, including the underlying skin and cartilage (Fig. 2f). If the tip is high, an additional segment is removed from the approximating border of the medial crus.

6. The medial crus, after being freed further (Fig. 2g) is then drawn outward for trimming. A section, usually about one-third the width of the crus, is removed up to the free border above, i.e., the entire length. Both skin and cartilage are included (Fig. 2h). This aids in shortening the nose and improves the contour of the tip.

7. Three fine black silk sutures are placed in site to unite the columellar skin and the skin remaining on the medial crus (Fig. 2j).

8. The exact procedure is carried out on the opposite side.

9. Light vaseline gauze packing is inserted into the nostrils to help maintain the cartilages in position.

10. Adhesive strips and dental molding (stent) compound lined with flannel are applied according to the method suggested by Aufricht.[3]

The apparent advantages of this method are:

1. The upper convex portion of the alar cartilages, which give an undesirable appearance to the tip, are removed completely.

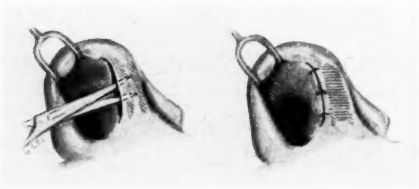

Fig. 3.—Correction of retracted columella. Incision, on one side only, along anterior border of medial crus and dissection of a pocket between the medial crura. Transplant placed in pocket. Three black silk sutures to close incision.

This approach may also be used for dorsal implants combined with columellar supports in saddle nose deformities by extending the incision along the lateral crus and undermining the skin over the nasal dorsum.

2. A complete skin lining is present over the upper half of the alar field, preventing scar contractures.

3. The lower field of the lateral crus and the complete medial crus are made readily accessible for modeling by pulling the freed cartilage forward from its bed. A proper wedge at the angle may then be removed, narrowing the width and lowering the height of the tip.

4. The medial crus is free for modeling. In the majority of cases a small section of the anterior rim of the medial crus and its attached skin is to be removed. This prevents a hanging nasal tip or "hooded" appearance of the forward projection of the tip.

5. If a hanging columella, which is caused by an excessive curve and enlargement of the medial crus, is present it can be corrected in this one procedure by removing a proportionately larger portion of the medial crus, which has already been exposed.

6. This method aids in preventing the removal of too great a portion of the septum in shortening the nose. The removal of the upper half of the alar cartilage, plus the section from the anterior rim of the medial crus, will in itself cause a moderate shortening of the nose so that removal of a smaller portion of the anterior border of the septum is required.

Correction of Retracted Columella and the Placing of Dorsal and Columellar Transplants for Saddle Nose. The methods most

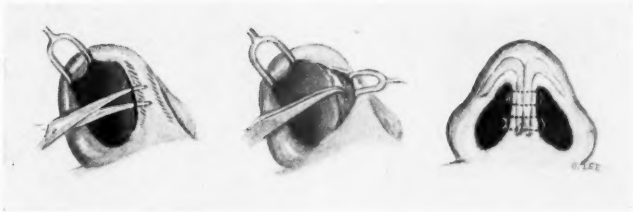

Fig. 4.—Correction of cleft tip of nose. Incision along anterior border of medial crus and cartilage dissected free. Procedure repeated on opposite side. Connective tissue between both crura removed and mattress sutures inserted to draw crura together. Original incision closed on each side with three fine black silk sutures.

commonly used for a retracted columella have been to place a cartilage transplant or strut between the septum and the columella. The membranous septum is completely or only partially incised, and the medial crura of the alar cartilages are dissected from behind to fashion a pocket to receive the transplant. The difficulties of this method are that immobilization of the strut is cumbersome and may not adequately fill out the retracted columella.

If one assumes that the hanging columella, which is the reverse of the retracted columella, is due to an excessive projection and enlargement of the medial crus, the proper location for the transplant would logically be in this position. I therefore do not incise the membranous septum at all but make an incision along one side of the anterior border of the medial crus for its entire length. The skin of the columella is then retracted and a pocket dissected between the medial crura and down to the anterior nasal spine. The transplant is placed in this pocket and a few small sutures are inserted between the skin of the columella and that of the medial crus (Fig. 3).

The transplant is thus fixed and cannot move because the membranous septum is intact and the cartilaginous septum lies rigidly behind it. The medial crura on both sides of the transplant hold it in a central position.

This method has proved successful in cases of retracted columella (Fig. 5) and also in many cases where support for the nasal tip, by a strut, was necessary.

This approach may be utilized for the insertion of dorsal implants in saddle nose deformities. The incision along the anterior

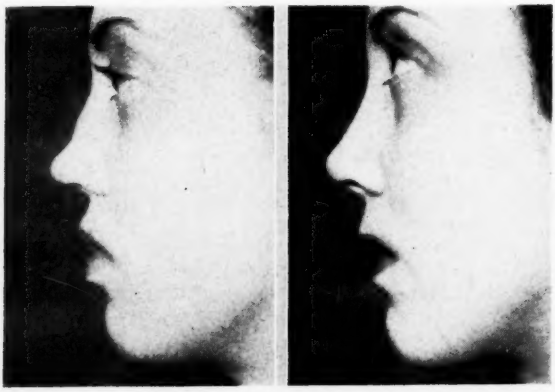

Fig. 5.—Nasal deformity with retracted columella. Representative case showing correction of nasal dorsum, alar cartilages and retraction of columella.

border of the medial crus is extended to include about one half of the lateral crus margin. Through this incision the skin and the periosteum over the midline of the nasal dorsum is undermined so that the cartilage transplant can be inserted.

The use of a cartilage strut (columellar support) combined with a dorsal implant is often necessary in saddle nose deformities when there is insufficient tip support. I have found this approach particularly advantageous for these cases because the nasal dorsum can be elevated for the dorsal implant and a space created behind the columella for the insertion of the strut all in the one procedure. Since one continuous space is created from the nasal dorsum down to the nasal spine, the columellar strut may be placed below the dorsal implant under direct vision. If necessary, a catgut suture can be used to unite the two pieces of cartilage. When a particularly rigid support for the nasal tip is needed, an L-shaped section of rib cartilage, combining the strut and the dorsal piece in one solid block, can be inserted.

Further advantages of this procedure are that the strut cannot move backward because the membranous septum is intact and the medial crus on either side holds it in a central position. To further insure the position of the strut the anterior pocket is dissected down to the nasal spine of the maxilla and a mattress suture is inserted between the anterior and the posterior border of the medial crura.

Cleft Tip of Nose. Frequently a split or cleft tip of the alar cartilages is encountered. Two methods for correction are usually employed. One is the method of dissection from behind, which includes incising the membranous septum and then dissecting forward between the two medial crura followed by the insertion of mattress sutures. The other method consists of an incision of the columella externally in the midline, dissecting the two crura and removing the connective tissue between them, then uniting the cartilages by mattress sutures. Removal from behind is often inadequate, and correction from the front results in an external scar.

A method which I have employed to great advantage is to incise the anterior border of the medial crus up to the lateral crus, including a small portion of the lateral crus past the angle. This is done on each side. The medial crura are then visible. The cartilages are then dissected free and any tissue which is present between the two crura is removed under direct vision. The cartilages are then drawn together by one or two mattress sutures and the skin margins between the columella and the anterior border of the medial crus are united as previously described (Fig. 4).

Elongated Columella. At times, in an excessively long or a very prominent nose when it is necessary to lower the profile closer to the face, a kinking or bending of the columella will appear. This is due to excessive length of the columellar skin and medial crura. Removal of a section of the medial crus (transverse section) on each side is not sufficient to relieve this kink. It is therefore necessary to remove a section of columellar skin and cartilage together.

This procedure is carried out at the junction of the columella and the upper lip so the scar is not so plainly visible. A complete transverse section of both skin and cartilage is removed, the size depending upon the amount necessary to relieve the kink. Fine black silk on an atraumatic needle is used to close the incision.

30 North Michigan Avenue.

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XLIX

CLEFT PALATE

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The history of attempts to correct cleft palate goes back to 1766 when Le Mormir reported his results. Eustachi in 1779 reported results, and in 1783 he wrote what seems to Dorrance[1] to have been the first description of a technique for closure of cleft palate.

The fundamentals of corrective measures for cleft lip and palate have remained the same for these past 50 years. However, variations, improvements and undesirable methods have naturally been introduced by many during the last half century. In selecting the method for his adoption, the young surgeon has a variety from which to choose and should select a satisfactory one which is acceptable to the majority of competent men and which he in particular can do best. Later he can add his own variations.

This paper deals with clefts of the soft and hard palates only and not the clefts of the alveolar arch and the lip. Our present-day treatment of these clefts is indeed a great advance over the earlier surgical procedures. Improved operating-room equipment to facilitate the work, present-day excellence of anesthesia, well-trained assistants probably outweigh any great changes in surgical ideas in so far as the good of the patient is concerned.

In our practice, great attention is given the preoperative examination and preparation of the patient. Since the great majority of these patients are operated on while very young, we do not have more than a few leads as to the fitness of these young subjects for this rather radical and often extensive operation. Therefore, we feel that the following preoperative observation of each patient is important.

PREOPERATIVE ORDERS

(These orders should be read by the resident and the interne before seeing any patient.)

(The nurse in charge should be thoroughly familiar with all of these orders.)

Read before the Sixty-Seventh Annual Meeting of the American Laryngological Association, Chicago, Ill., May 29, 1946.

1. A careful physical examination is to be done, including ears, nose and throat, teeth or gingiva, and a report is to be written and signed by the interne or the resident on admission.

2. The interne is to question the family carefully as to recent illnesses or exposure to communicable disease or infection and as to family tendencies to disease; later the family should be questioned by the nurse in charge because they may forget or refuse to tell the doctor of recent exposure to infectious diseases, family tendencies toward such diseases as hemophilia or others that would contraindicate operation.

3. If the child is fretful after admission, give a hypnotic, in dosage according to age.

4. No patient will be operated on with acute inflammation of the upper or lower respiratory tract or ears. The patient should be examined 12 hours before operation as the patient may show signs of infection after the examination has been done on admission.

5. A throat culture should be made on admission, to be repeated 24 hours before the operation. The following laboratory work is to be done on all patients:

Kahn test Complete blood count Hemoglobin determination Coagulation and bleeding time determination Urinalysis Vaginal swabs on all females, taken by the nurse in charge. X-ray study of thymus on all children six years of age or under.	Reports of these tests are to be on the chart within 24 hours after admission.

6. Give soda bicarbonate (or other alkali) in a sufficient amount 24 hours before operation to produce alkaline urine, together with 1 cc. of vitamin K.

7. Give adrenal cortex hypodermically, 1 cc. the night before and 1 cc. the morning of operation.

8. Give adequate fluids (water and orange juice) in amounts according to age—up to six hours before operation. GIVE NOTHING AFTER. Do not waken. Caution relatives not to feed or give fluids to the patient.

9. Take the patient's temperature one-half hour before operation. IMPORTANT.

10. Before sending the patient to the operating room, cleanse the operative area and the nostrils with boric acid swabs.

11. Medication preliminary to operation:

    1. Codein is to be given only *on order of the surgeon.*
       (The use of atropine has been absolutely discontinued.)
    2. An unusually restless or active patient two years of age or under should always have a hypnotic the night before and the morning of the operation.
    3. Children two years of age or older should have a hypnotic the night before and the morning of the operation. No opium is to be given except on order of the surgeon.

12. Keep the family from going to the operating room.

13. Send the infant or the child to the operating room in a gown and a blanket—no diaper. Be sure the colon and the bladder are empty. Send the patient to the operating room only when the anesthetic is to be started.

14. No infant is to be operated on:

(a) Unless modified cow's milk, mother's milk or other satisfactory formula has been provided. (Ask the surgeon.)

(b) Unless the stools are normal.

(c) Unless he is gaining weight. (Weigh daily.)

(d) If there is regurgitation of feedings.

(e) Unless he "seems good" to the pediatric nurse.

(f) Unless instructions 1, 2, 4, 5 are satisfactory to the surgeon.

15. Preoperative iodine or thyroid therapy. IMPORTANT.

The surgeon is to be questioned regarding this by the nurse in charge on admission of the patient.

According to age, a suitable form of iodine in maximum dosage is given from the time of admission.

Adults: Lugol, 10 ot 15 drops a day.

Children (from 4 to 6 years of age): Iodide of Iron or Syrup of Hydriodic Acid, one-half to one teaspoonful a day (If necessary, Oridine—chocolate-coated tablets).

Babies: Syrup of Hydriodic Acid, 10 to 15 drops a day.

All patients: Thyroid therapy—on order. Ask the surgeon. Thyroglobin. (Each drop contains 1/15 gr. thyroid extract.) Thyroid extract, for older patients.

In our experience, a statement by the pediatrician that the patient is in good condition for operation is not always sufficient. Too often pediatricians fail to realize that the child is to undergo a serious operation, when all body functions should be as nearly normal as possible. In the repair of a cleft of the hard palate in a young child where the blood supply to the flaps is not too extensive, a good quality of blood is most important if primary union is to be expected.

A carefully made, complete blood count and satisfactory information on the status of the thymus gland are our leading sources of information for these patients aside, of course, from a good report on the other vital organs. A nearly normal balance in the hemoglobin, red cell and lymphocyte count is of greatest importance in this operation since the flaps are stiff and rigid and the blood supply not nearly so adequate as in the soft palate and the lip. We have found by sufficient experience that any child with a high lymphocyte count is a dangerous risk and subject to severe physiological reactions.

One hour before operation the patient is given a hypnotic, usually nembutal; no atropine or narcotic.

Ether-oxygen vapor is the preferred anesthetic except in adults or large children, when the anesthetist may change this routine.

We prefer the Brophy mouth gag with ether tube attached. Instead of the bright nickel-plated ones, we have had them made of black nickel plate on the outside to reduce the glare. The inside is bright nickel for reflection within the mouth.

A No. 1 plain catgut suture is placed through the tip of the tongue for retraction.

The operation is begun by an incision along the extent of the cleft border where the oral and nasal mucous membranes join. This is done with the MacKenty half-circle knife. The elevation of the soft tissues and periosteum of the hard palate from the horizontal plates of the superior maxillary bone and the palatine bone is begun and continued with Brophy elevators. Some of these we have changed to suit our own personal likes. This must be carefully done. In many instances there are deep crevices in the soft tissues of the hard palate caused by deep attachment to the highly deflected horizontal plate. Tearing or mutilation of the apex of this crevice produces subsequent fibrosis in the newly formed palate. We make no attempt to fracture and include the horizontal bony plate in the palatal flaps as some operators do. We feel that the reattachment of the flaps to the plates and the subsequent contraction of the flaps bring these plates to a good position. The detachment of the flap extends toward the alveolar process, as far as necessary in each individual instance to secure good approximation.

Incisions along the lateral borders of the palate are never made unless necessary to secure adequate approximation of the medial borders of the cleft. Occasionally this step is necessary but only in a wide bilateral cleft of the hard palate and in some wide single ones. We feel that this, as a routine procedure, is not at all necessary and is to be condemned. The chief purpose of this paper is to establish this fact and encourage operators who begin the elevation of the flaps from the area of the alveolar arch instead of from the borders of the cleft to reconsider the Brophy technique.

The next step is the detachment of the hard and soft palate from the horizontal plates of the palate bones. This step again requires care not to rupture the oral surface and not, if possible, to break structures emerging from the posterior palatine canal. There is an excellent illustration by New on page 139 of Brophy's[2] book, "Cleft Lip and Palate."

A description of the muscle attachment and important muscles is given to Brophy[2]: "The muscles concerned in this attachment and the subsequent contractions on the suture line of the closed velum or soft palate are: Commencing with the mesial line, one can dissect out the attachment of five muscles, each of which is, of course, duplicated on the opposite side and each of which has such relations to a mesial line that, in case of cleft or split, it serves more or less to draw the parts posterolaterally. These muscles, mentioned in order of their significance to such lateral displacement, and consequently, in their relation to the operation of staphylorrhaphy, are the tensor palati, palato-glossus, levator palati, palato-pharyngeus and azygos uvulae. Of all of these structures the tensor palati plays the most important part and it is, therefore, entitled to the first consideration. The muscle arises from the scaphoid fossa, at the root of the internal pterygoid plate, from the anterior surface of the Eustachian tube and from the spinus process of the sphenoid bone. If the student carry his finger in his own mouth back to the wisdom-tooth of the superior jaw and let it drop over and back of this organ, it will fall on the tuberosity of the maxillary bone; carry it about half an inch farther back and it will come to a second prominence; this is the hamular process of the pterygoid plate of the sphenoid bone. The tensor palati muscle descends from the origin of which we have just informed ourselves, and meeting this hamular process, it winds, as a tendon, around it and then by a fan-like expansion spreads itself into the substance of the soft palate. Its action is evident: it expands the palate laterally and dilates the pharyngeal orifice of the Eustachian tube."

Next the entire border of each cleft is pared sufficiently to secure good approximation without tension and an adequate surface for good blood supply.

Next, two pilot sutures of No. 1 moistened plain catgut are introduced through the body of the soft palate. These are to draw through each body of the cleft muscular soft palate No. 20 gauge silver wire and to be united over lead plates as stay sutures to overcome the contraction of muscles which will inevitably tear out the suture line. You will note that the *only* areas for fibrosis in the muscle body is a perforation for a 20-gauge silver wire. No lateral incisions and no section of the muscle have been done. Neither have any wide incisions around the periphery of the soft palate been made. The abstinence from these procedures reduces fibrosis and contraction, which can be very extensive.

The cleft borders are then approximated. Suture material is horsehair and steel or tantalum wire, usually both. The posterior or nasopharyngeal mucosa is closed with horsehair up to the attachment to the aponeurosis. Lastly, when the entire palate cleft has been closed, the silver stay wires are united over the plates, and no tension is put on these sutures to draw the cleft borders together as this will result in necrosis.

This procedure, when it can be followed out, gives good speech and a nearly normal-looking palate. The functional success of any cleft palate technique should be evaluated when the patient has attained full growth and *not* six months after operation.

Postoperative care is given. It will be noticed that these orders, as well as the preoperative orders, apply to all cleft lip and palate patients.

POSTOPERATIVE ORDERS

(For babies and children—special orders for adults.)

Upon the patient's return from the operating room:

1. See that the patient has dry clothing; wrap him in a dry warm sheet and a warm blanket and use hot water bottles. HAVE A WARM BED. Do not chill the patient in changing his garments or wrappings. Put on wrist cuffs or restrain hands AT ONCE.

2. Watch respiration, i.e., see that the patient has easy free breathing especially if he has had a cleft palate operation. Call the interne if necessary.

3. Give saline glucose, by clysis or intravenously, with ascorbic acid (100 mg. for babies and from 500 to 1000 mg. for adults). Adrenal cortex is given and repeated if needed.

4. See that freshly prepared 6% solution of sterile glucose is ready *when the patient is returned from the operating room.* Have ready a sterile bottle (2-4 oz.), with sterile rubber covered medicine dropper tip, in hot water container for glucose solution. When the patient reacts and can swallow, give glucose solution (warm) carefully p.r.n., ad lib. by mouth to quiet and for 12 hours thereafter.

5. Feeding is to be ordered by the pediatrician. Have orders and formula established BEFORE the operation.

6. If the patient shows signs of shock or unnatural postoperative reactions, call the interne and surgeon. DO NOT WAIT.

7. If the patient is not quieted by glucose and is unusually restless and crying, give codein by hypodermic (by the interne's orders) for three doses. After that, the needed dose may be repeated every four hours if required. *Never give any narcotic until the effect of the anesthetic has passed off completely.* If it has been found by test dose of hypnotic that the patient reacts favorably to a hypnotic, use this in repeat doses in preference to codein or other narcotic. However, codein may be necessary if a hypnotic will not be retained.

8. After each feeding (formula) give sterile water to cleanse the mouth. The liquid formula should be started in part 24 hours after operation; sterile glucose solution should be given up to that time. Rapidly increase to full liquid formula.

9. Give an enema the night of operation day. After that give a daily enema unless or until bowel movement is normal. Much must be left here to the discretion of the carefully observant pediatric nurse in charge. Sufficient cleansing is to be done but not overdone.

10. Ask the surgeon about postoperative treatment of wounds.

11. Watch that relatives do not feed the patient.

12. *Never give solid food or semi-solid food until so ordered by the surgeon.*

13. Removal of sutures: The nurse in charge is to ask the surgeon about this soon after the operation.

Lip: Superficial skin sutures should be removed on the fourth day—by the interne or pediatric nurse in charge but never by a student nurse. Deep sutures are to be removed later. *All sutures should be out in ten days.*

Palate: Suture removal is to be directed by the surgeon. If plates are in, watch closely from three days after operation that they are not too tight (indicated by swelling of the tissue).

Any lip suture, loose or superficial, should be removed when not supporting tissue closure. Also, any suture which seems to be promoting infection should be removed and the surgeon notified.

Palate sutures showing white or necrotic areas should be reported to the surgeon.

14. Notification of relatives: Signed permission for surgery on minors must be obtained from nearest relatives. An understanding between the nurse in charge and relatives must be in effect. A baby may not be in condition on the day set for operation but may be two days later. She must arrange the notification and have permission to *operate when the patient is in condition.*

15. The lip bridge in cleft lip operations remains in position for eight days; it must be tight and adjusted daily. The lip bridge is to be removed *only* on order of the surgeon.

16. Postoperative sedatives are to be given just as preoperative. Hypnotics are preferred, but if not feasible to administer, give codein or other narcotics *on order.* I do not want an infant or older child to have an unnecessary restless or excited postoperative period. Some are in pain, some nervous, some frightened and homesick—some are spoiled; be as sure as you can of the condition.

17. Thyroid medication, if prescribed before operation, is to be continued, but with the surgeon's attention called to it.

18. If at any time the condition of the patient does not "seem good" to the pediatric nurse experienced in the care of general surgical as well as cleft palate and lip patients, she is to call the surgeon in charge.

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RHINOSPORIDIOSIS

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Rhinosporidiosis is a tropical disease about which little is known in the United States. Since we are often called in consultation in difficult or obscure cases, we should know the difference between ordinary nasal polypi and polypi in rhinosporidiosis.

The disease has been reported in East India, Argentina and the United States. The likelihood of more cases being brought back from East India since the close of the second World War gives added importance to a disease which has so far been rare in the United States. Three cases have been reported from Argentina and at least 25 from East India since the first case was reported in East India in 1903. Seeber reported this disease in Argentina in 1900 in Buenos Aires. There have been three cases in the United States according to Weller and Riker.[2] Ashworth[1] has given a complete history of the disease. Shrewsbury[3] has also shown excellent plates of the Rhinosporidium seeberi on artificial media.

The cause of rhinosporidiosis is the rhinosporidium seeberi. Very large, almost round spores are formed. These should be easy to recognize. The sporidium belongs to the lower fungi in the suborder chytridinease.

The gross appearance of the growth is much like that of an ordinary nasal polyp. At least those from the nose are. The tumor is more vascular than a nasal polyp and the surface is often rough. The growths are friable and bleed easily. They are usually pedunculated. The rhinosporidium can be seen by the naked eye as white, pin-point spots. This is "a fungous infection of the mucous membrane of the nose, with polypi, tumors of the cheek, conjunctivae, lacrymal sac, and uvula. It occurs in Argentina, India, Ceylon *and the U.S.A.* It is due to the Rhinosporidium seeberi, a yeast or phycomycete, spherical organism in polypoid growth; oval spheres (6 u) with chitinous envelope, vacuolated cytoplasm and vesicular nucleus.

Read at the Sixty-Seventh Annual Meeting of the American Laryngological Association, Chicago, Ill., May 29, 1946.

When fully grown, the sporangium measures 0.25—3 mm., it bursts and discharges spores which spread to connective tissue via the lymph channels. It can be cultivated with difficulty on Sabourands' medium."[4]

The polypi are seen to be edematous and have a connective tissue stroma when examined with the lower powers of the microscope. There are many infiltrating cells, chiefly lymphocytes and plasma cells. There are a few polynuclear and polymorphonuclear leucocytes. Very few eosinophiles have been found.

Regarding the symptomatology, there may be more or less complete nasal obstruction on one or both sides of the nose. Often there is a single polyp attached to the mucous membrane of the nose over the quadrangular cartilage. The attachment may be rather high or it may be at the anterior end of the inferior turbinate and to the turbinate. There is some nasal discharge, but no pain. All of the cases to date are in males of ages from 10 to 60. There are more than 40 cases reported to date. The polypi recur for a few years following removal, but not indefinitely. Upon removal these bleed freely.

In the diagnosis of rhinosporidiosis the microscopic examination, with a culture of the fungus, will show the difference from an ordinary nasal polyp. All nasal polypi should be examined microscopically. Those caused by the rhinosporidium are more vascular and consequently bleed more when removed. Many of these have been reported as a single polyp attached to the septum or the anterior end of the inferior turbinate.

In treatment surgical removal with a snare, under local anesthesia, is best. However, all of the pedicle should be removed and the base cauterized with the galvanocautery or desiccated by surgical diathermy. The polypi whether single or multiple tend to recur unless the base is cauterized. Recurrence may take place from another part of the nasal mucosa. Care should be exercised to avoid rupture of the polypi and infection of the surrounding nasal mucosa from the infected fluid contained in the polyp. The intravenous administration of tartaremetic has been used with some advantage.

Ashworth[1] reported a case seen with Dr. Logan Turner in Edinburgh. The patient was Dr. M., a graduate in medicine, who was born in April 1891 at Ernakulam, the capital of the State of Cochin, on the southwest coast of India, and he lived there until he was about 20 years of age. In late 1910 or early 1911 he became aware of a growth in his nose extending into the nasopharynx. He

was able to see the lower end of the growth, below the free edge of the soft palate, when he used a mirror to view the oral cavity. Before swallowing he had to force the polyp up into the nasopharynx. The growth was removed about a year later in Cochin. It returned and had to be removed a second time in eight or nine months. One year later the patient was in Madras. He felt a growth in the left side of the nose farther forward than the first. This bled occasionally. In 1913 the patient went to Edinburgh. He was annoyed by the bleeding and profuse viscid mucous discharge. In 1914 the growth was removed by Dr. Turner. In May 1917 Dr. Turner again removed growths, which was the sixth time these had been excised. At this time Ashworth[1] examined the specimens for parasites and he found the rhinosporidium. Dr. Logan Turner removed polypi on seven subsequent occasions.

In June 1917 the tumor was 15 x 10 x 7 mm. in size. Another removed a few days later was 33 x 10 x 7 mm. The polypi had a mammillated appearance and contained many large parasites. The polypi were redder than ordinary nasal polypi.

On May 12, 1921, Dr. Logan Turner did a Rouge operation. He tried to remove the growth completely from its attachment to the nasal septum over an area 20 x 14 mm. in size. He also removed tissue from the ethmoidal region and from near the antrum.

In conclusion the important points to be remembered about the disease are as follows:

1. The disease is rare even in tropical and semitropical countries.

2. The lesions are usually confined to the nasal cavities.

3. These resemble polypi, but are redder; are often attached to the septum or inferior turbinate; bleed easily. The fungus can be seen with the naked eye as small white or gray dots.

PHYSICIANS BUILDING.

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LI

TONSILLECTOMY AND POLIOMYELITIS

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Attention has been focused in recent years on the relationship of tonsillectomy to poliomyelitis. As one surveys the literature,[1-11] it becomes increasingly evident that some relationship may exist. One cannot deny the numerous, tragic, acute infantile paralysis attacks following recent surgical removal of tonsils. Such evidence cannot be brushed aside as unimportant.

Despite the general acceptance of such a relationship, it is still a matter of major interest as to whether the relationship is causal or coincidental. The evidence thus far submitted fails to answer the question.

The etiology and the epidemiology of anterior poliomyelitis are the greatest unknown factors in any statistical relationship as to the relative infectivity or incidence of this disease.

Furthermore, the mode of transmission, the incubation period, the portal of entry and the habitat of the causative virus are not known.

In dealing with such a dramatic and treacherous disease as poliomyelitis, capable of arousing unreasonable panic in the general population, it is wise to consider carefully all possible precautions and utilize them. It is that consideration perhaps, more than the scientific evidence submitted, that has led to a general adoption of the practice of postponing tonsillectomies during poliomyelitis epidemics.

There is some evidence of an experimental nature which tends to make one believe that there is a causal relationship. Sabin[12] showed that injection of the virus into the tonsillopharyngeal region of monkeys produced poliomyelitis. In 13 of the 16 monkeys thus treated, she found histological evidence of involvement of the nuclei of the cranial nerves, and concluded from these experiments that the poliomyelitis virus entered and reached the nuclei of the cranial

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nerves through the peripheral nerves supplying the tonsillopharyngeal area.

Yet when she attempted to produce poliomyelitis in monkeys by painting the virus over the operated areas of recently removed tonsils, she was unable to produce the infection. Two other investigators, Toomey and Krill,[13] removed the tonsils of six monkeys and then flooded the operated regions with ten per cent virus suspension for five days. Again, these monkeys resisted experimental infection.

Because of many years of experimentation upon anterior poliomyelitis, the nasal and pharyngeal mucosae have been considered the portal of entry or nidus of the causative virus. Notably among these works are the studies of Flexner, Lewis, Clark, Harrington, Sabin, Olitsky, Lennette, Hudson and Armstrong. On the other hand, other workers have provided evidence against the upper respiratory mucosa as being the only pathway of infection for some strains of infantile paralysis virus.[30, 34, 37] Amos and Taylor[24] have demonstrated poliomyelitis virus neutralizing agents in the nasal pharyngeal washings of man. These agents have properties of a lysozyme and appeared to diminish in quantity in certain inflammatory diseases of the upper respiratory tract. Burnet, Lush and Jackson[26] have reported anti-virus substances from human secretions, particularly for the influenza and herpetic viruses, as well as some neutralizing agents against poliomyelitis virus. These findings suggest that the nasal secretions contain important viricidal agents. The work of many investigators indicates that the nasal secretions are the source of bacteriolytic lysozymes, which is probably true in the case of virus agents.

The nasal secretions, normally, are so scant that they are not a very practical source of lysozymes such as is saliva or tears. In passing it is noteworthy that these natural bacteriolytic agents led to the search and discovery of other agents in nature, such as penicillin.

When local or general circumstances arise which lower the resistance or decrease the amount of the lysozymes, pathogenic bacteria then can be implanted upon the nasopharyngeal mucosa. This applies also to the viruses. Possibly, if tonsillectomy has any effect, it may be by lowering the resistance of the bodily functions, and not due to the local operative wound.

Recently, Holtman of the University of Tennessee reported that the effect of summer heat on the body chemistry may increase sus-

ceptibility to poliomyelitis. He has shown by experiments on mice that the ones maintained in summer heat temperature began showing symptoms and dying as early as the fifth day after inoculation with poliomyelitis virus, whereas the ones kept in a temperature around 55° F. showed no symptoms until the eleventh or thirteenth day.

He believes his findings with mice mean that the rapid growth of the poliomyelitis virus in the body and the resulting symptoms of the disease are dependent upon a disturbance of the normal metabolism of the body brought about by changes in temperature.

The most significant evidence on the causal relationship of tonsillectomy and poliomyelitis is a recent paper by Faber and Silverberg.[14] Here is evidence from histologic study of eight patients who died of acute poliomyelitis. The study was an attempt to determine whether the virus enters the mucous membranes through superficial nerve fibers, infects the neurons in peripheral ganglia, and then proceeds into the central nervous system. Based on the histological evidence from these eight cases, Faber comes to the conclusion that the pharynx appears to be an especially favorable site for the primary penetration of the virus into the body.

Referring directly to the pathway of infection following adenotonsillectomy, Faber has this to say: "The motor nerves of the pharyngeal muscles probably serve as portals of entry in poliomyelitis following adenotonsillectomy, with the virus ascending directly to the nucleus ambiguus and causing pharyngeal paralysis. The process, resulting as it does from gross trauma, must be regarded as exceptional."

This histological evidence, significant as it is, is based on the findings in merely eight cases. It is still insufficient to prove adequately a causal relationship.

It is wise to remember that this evidence, both positive and negative, is experimental. How closely can one translate the findings of monkey experiments and relate them to human infection? In a disease that is known in man alone, and capable of being produced artificially only in a limited number of animals, the results of such experiments must be closely scrutinized before being applied directly to the human disease process. There have been numerous occasions in the past where too much emphasis has been placed on the reliability of evidence accruing from animal experiments in poliomyelitis. The attempts to produce active immunity in humans by the use of vac-

cines, and the endeavor to block ingress of the virus into the human body by the use of zinc sulphate nasal sprays, must be remembered. Both these methods produced remarkably effective results in the laboratories, but the principle established in these animal experiments did not hold true in human trials.

There are several considerations to be aware of before one can reach a definite conclusion. There is a general belief at present that the poliomyelitis virus is widely disseminated in the population during epidemic periods. From many sources we have been informed of the large number of persons who harbor the virus with few if any symptoms. This fact poses an interesting question. Why can many individuals be host to the virus and show only mild or no clinical signs of the disease? Immunity is not a sufficient answer, for the process of immunity in regard to poliomyelitis is still conjectural. While it is true that special neutralizing tests have been developed to demonstrate immune bodies in the human blood stream, it is likewise true that recognized authorities place but little credence in the presence of such antibodies as a factor in resistance of the individual to clinical attack. Humoral antibodies have no importance in a disease definitely neurotropic. The poliomyelitis virus is a total parasite with an affinity for specific neural tissue. Resistance is of a specific cellular, neurotropic nature. Medical science has not yet been able to develop methods to demonstrate such cellular immunity. The reason for such lack of symptoms in those harboring the virus can only be surmised. Inasmuch as the presence of the virus in these carriers has been demonstrated on repeated occasions, it suggests multiplication of the virus within the host. Growth of the virus, we know, can occur only in neural tissue. Therefore, even this benign state of infection must represent a neurological invasion of a degree sufficient to permit the growth of the virus, but insufficient to produce clinical symptoms. To explain this phenomenon, it has been suggested that an equilibrium has been established between the host and the virus which holds in check further inroads of the parasitic organism.

Reasoning further from this assumption, it is logical to assume that any occurrence disturbing that intimate and delicate balance might accelerate the virus invasion, thereby producing sufficient pathologic conditions to provoke severe clinical symptoms.

To determine the role of tonsillectomy in a causal relationship to acute poliomyelitis, one must also consider the attendant surgical shock and the effects of anesthesia. These factors, regardless of the

nature or site of operation, might be sufficient to disturb the equilibrium of host and virus. General anesthetics are capable of producing anoxia of the central nervous system.[15 16] It has been similarly established that the oxygen requirement of the central nervous system is approximately 30 times that of other tissue of the body.[17] Anesthesia, through the production of anoxia, might in itself be sufficient cause for disturbance of the host-virus relationship and capable of precipitating severe attacks of infantile paralysis.

Tonsillectomy is the one operative procedure most common to all children. In the age groups most susceptible to poliomyelitis (2 to 9 years) tonsillectomy is the most frequently performed surgical procedure. Are we putting too much emphasis on the site and nature of the operation and giving insufficient regard to the role of surgical shock and anesthesia?

There are yet to be determined many facts concerning poliomyelitis before definitely incriminating tonsillectomy in the etiology of bulbar and bulbospinal forms of poliomyelitis. The means of transmission is still a matter of conjecture. No carrier other than man has been found. Despite the occasional evidence incriminating flies and virus-contaminated sewage, the disease has occurred in areas free from flies and has not followed the characteristics of waterborne infections. The portal of entry has not yet been determined. The nose, pharynx and alimentary tract have their adherents; but the scientific evidence to establish any or all of these portals is still obscure. The neural pathways of infection have not been completely worked out. There is as much evidence for an afferent spread of the disease, as there is for the efferent theory.

It is agreed by most authorities that lack of uniformity in official reporting of poliomyelitis makes accurate interpretation of statistical evidence almost impossible. Many states record only severely paralyzed cases; other states report the mild and abortive types as well. Pertinent histories seldom accompany the recording of the case.

Following the severe outbreaks of the disease in the eastern and southern states during 1944, the writer requested certain information from the State Departments of Health in those states where the disease was most prevalent.

The specific information requested was: (1) the number of cases of infantile paralysis; (2) a history of recent tonsillectomy. While it was possible to secure answers to the first question, none

of the officials were able to supply answers to the question of recent tonsillectomy among the recorded cases of poliomyelitis.

With ever-increasing attention being given to the possible relationship of tonsillectomy to poliomyelitis, such pertinent information gathered from many sources will help to clear the problem. It would be within the interest of our national nose and throat societies to request state departments of health to add a tonsillectomy history line to their official reports on poliomyelitis. Without this information, the results of statistical surveys will be inconclusive, regardless of the sincerity of the investigation.

Until these basic facts of the disease can be established, the relationship of tonsillectomy and poliomyelitis cannot be conclusive.

Recently, Dr. John R. Page surveyed the incidence of poliomyelitis following tonsillectomies performed at the Manhattan Eye, Ear and Throat Hospital during the years of 1937, 1939 and 1941 when the disease in New York City was above normal expectations. During the three year period, 27,849 tonsillectomies were performed. Cards were mailed to these individuals requesting detailed information on subsequent illnesses. Replies were received from 8,915 patients. There was but one case of poliomyelitis reported in the replies.

An extension of this survey was attempted by the writer for the months of July, August and September of 1942, 1943, 1944 and 1945. For the year 1944, when New York suffered the most severe epidemic in recent years, the months of October and November were added to the statistical survey. Cards were sent to 5,470 patients (in the 3-16 year age group), who underwent tonsillectomy at the height of the epidemics and replies were received from 2,289. There were three cases of poliomyelitis in this group. None of them were of the bulbar type and all three reported as mild cases.

Dr. Morely T. Smith, Director of Otolaryngology at Grasslands Hospital in Westchester County, was kind enough to send me a report on the number of acute poliomyelitis cases admitted to that institution during the severe epidemic of 1944. There were 104 cases admitted (in the 3 to 16 year age group). In this group there were 13 cases of the bulbar type. None of these patients had had tonsillectomies within two months of the onset of the disease.

Dr. Edward Whalen of Hartford, Conn., also sent me the figures as compiled by the Connecticut State Board of Health. In the year 1945 there were 10,000 tonsillectomies done in that state.

There were 214 cases of poliomyelitis reported, 12 of the bulbar type.

None of the patients having the bulbar type had had recent tonsillectomy.

There were three cases of mild poliomyelitis following the removal of tonsils just prior to the onset of illness.

Dr. Ben Shapiro, Chief Pediatrician at Mary Immaculate Hospital, Jamaica, New York, also sent me a report from that institution for the year 1944. There were 39 cases of poliomyelitis admitted, two of the bulbar type. One case, which was of the spinal type, followed recent removal of tonsils.

Aycock,[18] after surveying the literature in relation to this question, in a compact summary states: "From the data available, it is not entirely clear whether the tonsillectomy or the adenotonsillectomy is the associated factor."

Owing to the importance of this subject of tonsillectomy and poliomyelitis and since there is a diversity of opinion among physicians, the writer suggests a nationwide survey be carried out by the American Laryngological, Rhinological and Otological Society. It is proposed that each Section Chairman appoint one or more members from each state to gather pertinent information that would go to make up a statistical report to be returned to the Society at the next annual meeting in 1947. This information could be readily obtained from state and municipal health organizations and would entail but little labor on the part of the physician. Such a report, which would include the entire country, would show on the part of each state the number of cases of poliomyelitis, the number having had a recent, remote or no tonsillectomy, the type of infection and other pertinent information.

He believes that such a survey will show no causal relationship between tonsillectomy and poliomyelitis. A favorable report, countrywide in scope, will do much to allay the fears of the public which are now widespread.

SUMMARY

The mode of transmission and the portal of entry of the virus of poliomyelitis remain unknown. Without this knowledge we are forced to theorize on the relationship between tonsillectomy and poliomyelitis.

A statistical survey indicates that poliomyelitis is relatively infrequent following tonsillectomy. The study carried out at Manhattan Eye, Ear and Throat Hospital on 11,204 tonsillectomy patients over a seven-year period revealed but four cases of poliomyelitis following tonsillectomy. None were of the bulbar type.

The widespread alarm on the part of the public, and shared by doctors in some communities, is unfounded on the basis of our statistics.

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LII

TONSILLECTOMY AND POLIOMYELITIS*

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My first interest in this subject was prompted by two lines published in a circular letter to physicians from the Connecticut State Department of Health,[1] which I quote: "As poliomyelitis sometimes develops after tonsillectomy you may wish to delay operation in case you have any tonsil operations."

I thought it my duty to advise my hospital of this statement and I did so. This promptly created anxiety and the hospital immediately excluded patients for tonsil operations. A group of children awaiting tonsillectomies that day were admitted only after a complete understanding with the parents of the possibilities suggested by the above statement made in the Connecticut State Health bulletin.

One-quarter of the parents took their children home.

It seemed to me then, as it does now, that there was little reason for panic. Here was a disease, recognized for a hundred years. Here was an operation which had been, for over 30 years, routinely deferred to the summer months when it was convenient to do it. It was not my understanding that any very intimate relationship had been accepted between tonsillectomy and poliomyelitis. For a much longer time, of course, tonsillotomy and also the operations on the throat, nose, teeth, jaws, mouth and other portions of the respiratory and gastro-intestinal tracts had been done *with* respect to necessity and convenience, but *without* regard, so far as I knew, to the alleged risk mentioned above. I decided to ascertain what statistics might reveal.

I wrote several institutions of international repute whose literature on the subject of infantile paralysis is rather voluminous, only to be referred to the publications with which I was already familiar. I wrote several research centers and health departments, only to be advised (1) that pertinent statistics were not available and probably

*Adopted from a discussion of the previous paper "Tonsillectomy and Poliomyelitis" by Dr. Daniel S. Cunning read at the annual meeting of the American Laryngological, Rhinological and Otological Society, Chicago, Ill., May 28, 1946.

would not be for an indefinite period on account of the pressure of other matters, or (2) that studies were being made of certain records and that if the information I sought became available, I would be so advised.

Apparently this data has not been assembled.

I then decided to canvass my own state. Accordingly, on November 1, 1943, I wrote 42 Connecticut hospitals inquiring about the number of tonsil operations done in these hospitals during July, August, September and October of that year. I also wrote 90 otolaryngologists asking whether, to their knowledge, any cases of poliomyelitis had followed recent tonsillectomies during this period.

Replies from 41 hospitals disclosed that 6610 tonsil operations had been performed during these months (in 1943); that 19 of these hospitals had excluded tonsil operations during a part of these months; and that one case of poliomyelitis was known to have developed in this group.

Of the 19 hospitals which were closed to tonsil operations during the poliomyelitis season in 1943, 14 were not closed during the same period in 1944 and 1945. Four were closed.

I was unable to get a reply from the one remaining hospital.

I received many replies from both hospitals and physicians which commented favorably on my efforts in this study and in several confidence was expressed that statistics would not reveal any convincing evidence of a consistent association between tonsillectomy and poliomyelitis.

Only one reply stood out in contrast which stated, in part, that any statistics I might uncover would carry little weight with hospitals or boards of health. This letter also raised the question of a possible medicolegal complication which might develop under unfavorable circumstances.

From the Connecticut State Department of Health I was able to obtain the following data: 346 cases of poliomyelitis had been reported during July, August, September and October 1943.

Of these 180 case records were on file.

Among these 180 cases, 56 patients had been tonsillectomized; 49 had not: in the remaining 75 cases no data on this detail was included.

Of these 346 cases, 5 patients (including the one mentioned above) had been operated upon between May 9 and July 2 and had developed poliomyelitis within 10, 8, 12, 8 and 4 weeks respectively.

But this small number of case reports would not serve as a satisfactory guide for those parents who feared operative delay more than poliomyelitis and who asked that tonsil operations be done during the summer months as usual.

Confusion was also added by some hospitals being closed and some open to tonsil operations.

Likewise, since this situation would not serve as a guide for years to come, it became quite evident that I would have to turn to nation-wide clinical reports of the incidence of this disease as associated with tonsillectomy, and to national health figures instead of state statistics.

A paper on this subject was read before the Midwestern Section of the American Laryngological, Rhinological and Otological Society in 1944 by Dr. Robert E. Howard of Cincinnati. In this paper[2] he stated that 259 cases of poliomyelitis had been reported following recent tonsillectomy in the 33 years 1910 through 1943. Adding to this number the 5 cases reported to me by the Connecticut State Department of Health, 9 more reported in the literature[3] since Dr. Howard's paper was read, plus 1 additional case reported to me, by him, in a personal communication, the total number of cases reported becomes 274, over the 35-year period since tonsillectomy and poliomyelitis were first associated in the literature.

Dr. Howard stated that no case of poliomyelitis had been reported following recent tonsillectomy in any patient over 18 years of age. The United States Public Health Service records show that 25 per cent of tonsillectomies are done in patients over 18 years of age, and also that two million tonsillectomies are done in this country annually.

This would give a total of 70 million tonsillectomies during the 35-year period, or 1 case of poliomyelitis reported to each 255,474.

Deducting 25 per cent of this figure for the age group 18 and over reduces the basic figure of 70 million to 52½ million and the incidence of poliomyelitis to 1 case to each 191,605 tonsillectomies.

Dr. Howard further stated: "The relationship of poliomyelitis to tonsillectomy and adenoidectomy should be based on the number

of such operations performed during the months when the virus may be present in the throat either at the time of operation or afterwards."

A survey of the number of tonsillectomies done at the Bridgeport Hospital in 1942, the last year the normal figure was uninfluenced by hospital closure or fear on the part of the public, disclosed that 53 per cent of all tonsillectomies done during that year were done during the four months, July, August, September and October, the months generally accepted as the poliomyelitis season except, again according to Dr. Howard, "in California and Texas where polio starts generally in June and lasts through November in most years studied." Assuming that this 53 per cent figure is a fair national average for tonsillectomy done during the poliomyelitis months, this would reduce the basic figure to 27,825,000 and the incidence of poliomyelitis to 1 case to each 101,551 tonsillectomies.

I am advised by Dr. Richard O'B. Shea, Health Officer, City of Bridgeport, that the average poliomyelitis incidence in every-day life is 1 to 3250 population. Accepting this figure and assuming that all cases of poliomyelitis reported (274) occurred during the poliomyelitis months (although they did not), it appears that the incidence of poliomyelitis in everyday life is 31 times more frequent than it is after recent tonsillectomy, even during the poliomyelitis season.

A stimulating speculation might develop about what statistics would reveal if the incidence of poliomyelitis were cross-checked with that of other diseases and with each of the operations upon the nose, throat, teeth, jaws, mouth and throughout the respiratory and gastro-intestinal tracts.

In talking with one bacteriologist concerning this point he said, "you may as well have all surgery stopped during the polio season, for rest assured, whatever a patient has in his alimentary tract he also has on his skin."

There is today, as there always has been and probably always will be, resistance in some quarters to tonsillectomy performed at any time. It is accepted practice among practicians to advise the several routine immunizations against anticipated diseases as a prophylactic procedure but rarely is tonsillectomy advised for the same reason. Tonsillectomy, combined of course with adenoidectomy, in young patients and performed as a prophylactic operation for less serious conditions, more often effects a striking improvement in the patient's condition than when it is employed for curative purposes.

Needless to say, every effort should be made to settle the question whether tonsillectomy predisposes to infantile paralysis, and if so, whether it influences the type of disease. That is important for the welfare of many thousands of children and for the comfort of many anxious parents. To us laryngologists, the seriousness of these questions is obvious and requires no rhetoric.

The incidence of the bulbar type in the 274 cases of poliomyelitis reported to date is approximately 45 per cent as compared with a 20 per cent incidence in Bridgeport in 1943.

In my city we are being pressed for an opinion by the public and the opinions most often given are expressed by women's clubs, parent-teacher's and visiting nurses' associations, all of whom the public considers well informed.

The press likewise has become interested and, if not taking sides, seems willing to express an opinion, if only by inference.

The following editorial appeared in "The Bridgeport Telegram" in June 1944:

"POLIO IS OVERPLAYED"

"The action of Trenton, N. J., in putting all children up to 16 years of age under quarantine against Infantile Paralysis serves to call attention to the fact that thanks to the late President of the U. S. and the Warm Springs Foundation, this is the best-advertised disease that we have.

"In some ways it is the most over-advertised disease because it is far less prevalent and far less injurious than some other diseases which hardly get any attention at all.

"For one child crippled by Infantile Paralysis, for instance, at least half a dozen will be crippled by rheumatic fever, which is hardly known to the general public, but is far more prevalent.

"Furthermore the victims of an Infantile Paralysis attack may lose the use of a limb wholly or partially but remain otherwise sound and able to carry on in normal fashion while the victim of a rheumatic fever attack may be crippled in the heart. To be crippled in the heart is to be crippled all over and that is a real tragedy.

"It is good to know that a nationwide drive against Infantile Paralysis is being made. It is somewhat unfortunate, however, that such overemphasis should be placed upon this one disease.

"The sensational action of Trenton, N. J., which practically confines all children to their homes, is more of a publicity stunt than a sound scientific or logical measure. Perhaps in the postwar era we may resurvey this whole subject of disease and put the emphasis where it belongs."

I feel sure that if some, if not all, groups interested in this subject could see what laryngologists see contained in the thousands of

tonsils operated upon each year, pressure against tonsillectomy would be much reduced.

No one but the laryngologist is familiar with these local conditions and while the foul and putrid matter contained in thousands of tonsils may not all be dynamite, it is sufficiently impressive to make other attempts at improved body and mouth hygiene fade into insignificance by comparison.

Naturally, no one would do a tonsillectomy or any other non-emergency operation during an epidemic of any disease and the proper attention should be paid to the apparent increase in the percentage of bulbar cases reported in post-tonsillectomy patients. However, one can not state that in another statistical study of groups of cases as large as these reviewed here, that this percentage might not be modified or reversed. Not infrequently, in smaller epidemics in a given area, statistics will show a high percentage of bulbar cases. Then, in a larger epidemic the next year in the same area, the figures may lead to a distinctly different conclusion but the series from which I have quoted is a large one and the period covered is a third of a century.

Thirty-five years is a long time to wait for contrary evidence.

SUMMARY

274 cases of poliomyelitis following recent tonsillectomy have been reported during the last 35 years.

It is estimated that 70,000,000 tonsillectomies were done during this period.

It is estimated that 75 per cent of this number, or 52,500,000 were done on patients under 18 years of age, the susceptible period.

It is estimated that over half of these operations, approximately 24,000,000, were performed during the poliomyelitis months.

If these 274 cases of poliomyelitis had all occurred during the poliomyelitis months (which they did not) the incidence would be 1 case of poliomyelitis to approximately 100,000 recent tonsillectomies.

It is estimated that the everage annual incidence of poliomyelitis in everyday life is 1 to 3250 of population.

These figures show that the annual incidence of poliomyelitis in everyday life is 31 times greater than in the recently tonsillectomized population even during the poliomyelitis season.

144 GOLDEN HILL STREET.

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The Scientific Papers of the American Broncho-Esophagological Association

LIII

CATHETERIZING BRONCHOSCOPE AND ANGULATED BRONCHIAL FORCEPS

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Much difficulty has been encountered in the past in the catheterization, suction and irrigation of the upper and middle lobe bronchi of the lungs. For this purpose a Jackson-type bronchoscope of 8 or 9 mm. diameter with a smaller inner cannula anteriorly for the light and one posteriorly for the catheter together with side tubes for the anesthetic and suction has been devised. A No. 6 ureteral catheter passed through the special cannula of the bronchoscope posteriorly can be readily guided into the upper or middle lobe bronchi, and the necessary procedure carried out. This is made possible by a small, curved lip at the distal end of the special carrying cannula which diverts the ureteral catheter at a 60° angle into the bronchus. This is especially advantageous in bronchoscopic lavage and aspiration and is superior to the procedure I previously outlined in 1934.[1] It may also be used for the collection of bronchial secretions for sedimentation cell studies. The advantage of this instrument is the angulation of the catheter, made possible by the curved lip on the distal end of the cannula, which facilitates the passing of the catheter into the bronchus.

Difficulty in approaching the upper and middle lobe bronchi has frequently been encountered with the straight or slightly angular bronchoscopic forceps now in use, because of the angulation of the bronchi at their junction with the main stem bronchus. A similar principle has been applied in a new type bronchoscopic forceps which is a modification of the Chevalier Jackson straight ball forceps for globular objects and bronchial biopsy. These forceps are

50 cm. long and consist of perforated cups or rings of 4 mm. diameter and can be used for the removal of globular objects, small tumor or tissue biopsy. The distal end of the shaft is angulated vertically so that the grasping stylet can be diverted 60 degrees and extended 1.5 cm. into the lumen of the bronchus, thus making possible a better approach into the upper and middle lobe bronchi. The rigidity afforded by the angulated shaft and the stylet makes this instrument more favorable for accuracy and ease of manipulation than the flexible type of upper lobe biopsy forceps.

19 GARFIELD PLACE.

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LIV

MAGNETS FOR THE EXTRACTION OF FOREIGN BODIES FROM THE AIR AND FOOD PASSAGES

PAUL H. HOLINGER, M.D.

CHICAGO, ILLINOIS

Interest in the use of magnets to remove foreign bodies from the air and food passages appears to come in cycles. This is in part due to the fact that clinical use follows advances in engineering fields, although it may lag somewhat until we become aware of these advances. According to Equen[1] magnets were first used in attempts to remove magnetizable foreign bodies over 100 years ago. Jackson describes their use in his first textbook on bronchoscopy[2] but little real value could be attributed to these early instruments because of their inefficiency.

The development of Alnico V added impetus to this work and several reports in the literature give evidence that a definite place for this type of foreign body extraction exists. Silber, Kaplan and Epstein,[3] Equen,[4] and Tucker[5] have used Alnico V in one form or another for this work.

The magnets herein presented differ from those previously described only in the type of handle to which the magnet is attached. This difference is significant since because of it the entire technic of the foreign body extraction is changed. The magnets are rods of Alnico V, 5 to 7 cm. in length and 3 to 6 mm. in diameter. Each is brazed to a brass rod; the rod for the smaller magnets is 35 cm. in length, while that of the larger magnets is 50 cm. in length. The smallest magnets may be passed through an infant esophagoscope.[6] The larger magnets have a rounded tip which is blunt enough to permit safe blind passage into the stomach. This magnet is guided into the esophagus by exposing the hypopharynx with a laryngoscope. It has the obvious objection of danger of perforation associated with any blind instrumentation of the esophagus and should be used with proper precaution. The advantage of the rigid shaft magnet is that it allows complete control under biplane fluoroscopy and the magnet may be directed to the foreign body quickly and accurately in both planes with a minimum loss of time.

A disadvantage in the use of the permanent magnet in foreign body extraction is that once a magnet attaches itself to a foreign body there is no means of releasing the foreign body if that becomes necessary other than to allow the wall of the viscus to hold the object and pull the magnet away from it. This may be dangerous in certain pointed foreign bodies. This is overcome if the small rigid magnet is used through the esophagoscope since the magnetic field is destroyed and the foreign body released if the scope is advanced over the end of the magnet.

Considerable question exists regarding the indications for use of magnets in foreign body extraction. In our experience their use would appear to be justified in the single problem of removal of long, magnetizable objects, such as bobby pins and long nails, from the stomachs of small children. This procedure which was formerly long and tedious with open tube gastroscopy now usually takes less than a minute with the rigid magnet. These foreign bodies are, of course, potentially dangerous since they might become lodged in the duodenum. In adults their passage through the gastro-intestinal tract is so commonly seen that one questions whether interference is necessary if the patient can be kept under observation until the foreign body has passed. If the foreign body is lodged in the esophagus or bronchus, on the other hand, the more positive procedure of use of forceps under direct vision would seem a more expedient method of removal, especially so in cases of pointed foreign bodies. Ball bearings in the bronchus, however, unless they are too deeply embedded, might adhere to the magnet sufficiently to permit extraction.

Since the use of magnets in rubber or woven catheters had been described elsewhere,[3, 4] an attempt was made to duplicate results with such instruments and compare them with the results obtained with rigid magnets. The objection to the flexible catheter shaft magnets was that positive guidance could not be obtained and chance contact had to be depended upon. To obviate this a heavy horseshoe magnet was used on the anterior abdominal wall in conjunction with the flexible magnet in the stomach. The external magnet drew the foreign body and the flexible magnet to the same point on the anterior wall of the stomach to facilitate contact.

SUMMARY

1. Three Alnico V permanent magnets brazed to brass rods are presented for use in the extraction of magnetizable foreign bodies from the air and food passages.

2. The rigid magnet permits positive control in leading the magnet to the foreign body under biplane fluoroscopic guidance.

3. The use of a strong magnet on the abdominal wall facilitates contact of the flexible magnet and foreign body if the flexible magnet is preferred to the rigid shaft type.

4. The indications and contraindications for the use of magnets in the extraction of foreign bodies from the air and food passages is discussed.

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LV

APLASIA OF THE LUNG

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The positive diagnosis of a case of true aplasia of the lung by x-ray examination, bronchoscopy and bronchography in an infant eight months of age appears to be sufficiently rare to justify its presentation. Although the medical literature of the past seven years reveals an increasing number of reports of such a congenital anomaly, it is a fact that the great majority of these represent postmortem findings rather than positive antemortem diagnoses. Killingsworth and Hibbs[1] stated that a careful review of the literature up to the time of their report revealed only 37 authentic cases. VanLoon and Diamond[2] found but 40 genuine case on record. They reported a proved case of agenesis of the right lung and bronchus by bronchoscopy and bronchography in a child three and a half years of age. The diagnosis was suggested by an x-ray of the chest for a routine physical examination. Garber[3] suggested that the sum total of proved cases reported did not exceed 100. Olcott and Dooley[4] reported a case of complete absence of the right lung in an infant two months of age, diagnosed by physical examination, x-ray study and bronchoscopy.

The etiology appears to be obscure. Many theories have been advanced, the one usually accepted as the most plausible being that of Schwalbe[5] who ascribed, "agenesis of the lung to a developmental error of endogenous origin—faulty germ plasm as it were—primary in the pulmonary, vascular and respiratory systems." Such an explanation seems to fit in with the supposed embryonic development of the respiratory system as described by Arey:[6] "A groove-like evagination arises on the ventral side of the esophagus in the 3 mm. embryo. From the posterior ends of the groove two small lung buds grow out. Later in the development the predestined anlage of the trachea and esophagus becomes separated by a constriction interrupted at the cephalic end by the larynx which can be seen in the embryo at the end of the fifth week. Muscle fibres and cartilaginous rings differentiate from the surrounding mesenchyme by the end of the seventh week. In a later development of the lung buds hollow evaginations grow out into the envelope of connective tissue, enlarge

rapidly and branch to produce the true tree-like tubular system. On the fine terminal tubules arise small outgrowths which constitute the alveoli." The foregoing description of development suggests that the type of anomaly in which there is complete absence of a main stem bronchus must have had its origin very early in embryonic life and other types in a correspondingly later period.

Most authors have accepted Schneider's classification of agenesis of the lung as follows:

1. True aplasia of the lung and bronchus in which there is no trace of a bronchus.

2. Aplasia of the lung in which the bronchus is represented by a blind pouch or a nodule of cartilage and fibrous tissue.

3. Extreme hypoplasia of the lung in which the main bronchus is normal in size and shape and ends in a fleshy structure.

Various other congenital anomalies are frequently associated with aplasia of the lung, such as absence of the ipsolateral pulmonary vessels, absence of the diaphragm, anal stricture, esophagotracheal fistula, accessory thymus, hypertrophic thymus, hernia of the diaphragm on the affected side, absence of the vagus nerve. The trachea is often narrowed and is apt to contain supernumerary cartilaginous rings.

Absence of a lung does not forecast an early death. There have been cases reported in which the individuals were aged 58, 65 and 72 years, respectively; all died of causes unrelated to the agenesis of the lung. The symptoms are variable and in many cases are absent, the condition being discovered only accidentally when for some reason, such as a general physical examination, the subject has had an x-ray of the chest. Dyspnea, cyanosis and a failure to thrive may be noted in the very young. The external symmetry of the thorax is maintained in most cases of true aplasia of the lung and bronchus. Apparently filling of the potential space by the displaced mediastinal contents and a portion of the hypertrophied contralateral lung at an early stage of the development prevents contraction of the affected hemithorax. The heart and the mediastinal contents are displaced to the affected side and the apical impulse of the heart is pronounced. Dullness or flatness on the affected side is common but there may be resonance because of the hypertrophy and the emphysema of the remaining lung. The breath sounds may be absent or suppressed.

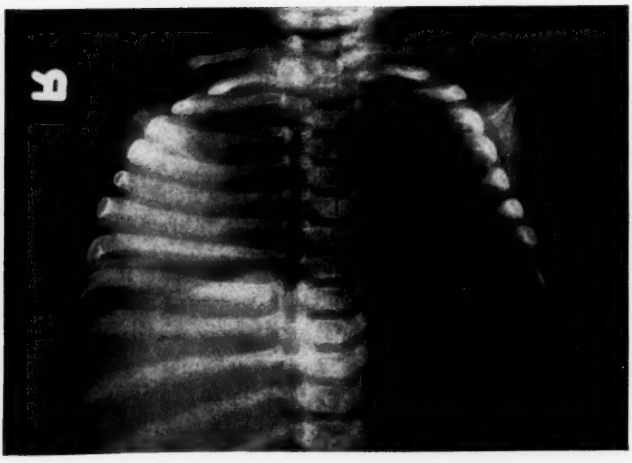

Fig. 1.—Roentgenogram made on date of admission showing the mediastinal structures displaced far to the right and a dense shadow in the lower portion of the right chest.

The usual roentgen interpretation is massive or fetal atelectasis.

Differential diagnosis in congenital absence of a lung is difficult. Pneumonia, hydrothorax, paralysis of the diaphragm, diaphragmatic hernia and foreign body in a bronchus must be considered.

REPORT OF A CASE

D. S., male, aged five months, was admitted to the Memorial Hospital, Wilmington, Delaware, October 27, 1945, because of slight dyspnea, developing during the course of a cold in the head.

The past personal history was essentially negative. There had been a normal birth; he was the twelfth child of the parents, all living. There was no history of previous illness.

Physical examination revealed a well-nourished, white infant with slight dyspnea apparently of lower respiratory origin. The thorax was symmetrical. There was no cyanosis. The temperature was normal. The heart sounds were heard upon the right side with the maximum intensity being to the right of the nipple. There was hyperresonance upon percussion over the left chest and dullness

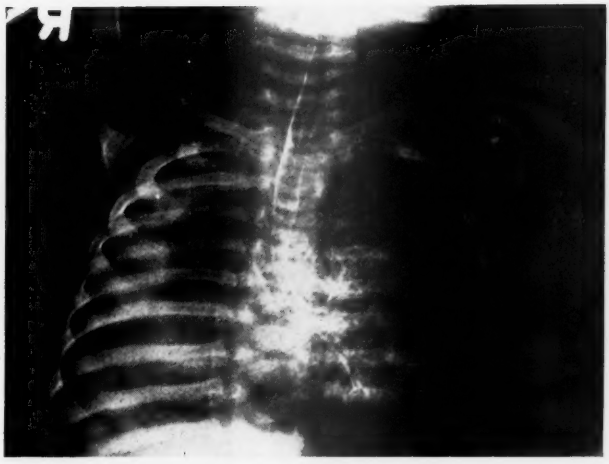

Fig. 2.—Bronchogram showing agenesis of right lung. There is no evidence of a bifurcation of the trachea.

throughout the right chest. The breath sounds on the left side were loud, but diminished to absent on the right side.

The urine was normal. The red count was 3,410,000. The white count was 7900 with a normal differential count. The hemoglobin was 10 gm.

The admitting diagnosis was massive atelectasis of the right lung.

X-ray examination upon the date of admission showed a dense shadow in the lower portion of the right chest. The mediastinal structures were displaced far to the right. The cardiac shadow and the right diaphragm could not be identified (Fig. 1). Barium was swallowed and showed the esophagus to be in the right chest. The interpretation of the roentgenologist was, "Aplasia of at least one of the lobes of the right lung, if not all of them or non-expansion. Inclined to think it to be aplasia." The roentgenologist consistently adhered to this first diagnosis upon each of the subsequent examinations. All of the films showed air in the upper right chest which was attributed to hypertrophied lung tissue of the left side which had crossed over to the right side.

Bronchoscopy performed with a 3.5 mm. bronchoscope November 15, 1945, revealed a comparatively normal mucosa with a rather large amount of mucous secretion. The trachea was deviated to the right. There was no evidence of a carina or a right bronchus. There was some dyspnea upon withdrawing the bronchoscope but this was not serious.

On January 23, 1946, iodized oil was introduced through a tracheal catheter without any complications. The report was as follows: "Bronchogram shows the trachea well outlined with oil. There is no evidence of a bifurcation. The trachea continues downward into what appears to be the left main stem bronchus. The left lung fills with oil and I believe there has been compensatory enlargement and shift of the left lung over to the right side.

Conclusion: This bronchogram indicates absence of a right main bronchus and lung due to right pulmonary agenesis."

The baby was discharged from the hospital January 28, 1946, but readmitted upon two occasions for a period of a few days for acute respiratory infections, each with complete recovery. At present, the child is convalescing from measles at the Contagious Hospital in Wilmington.

601 Delaware Avenue.

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LVI

AGENESIS OF THE RIGHT LUNG

WITH DEATH FOLLOWING ASPIRATION OF FOREIGN BODIES INTO THE LEFT LUNG

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LAKEWOOD, OHIO

In 1936, I[1] reported a series of unusual cases of foreign bodies in the food and air passages, which included one instance of agenesis of the lung. Since this report is not readily available to students of this condition and since it has unusual interest for bronchoscopists, it seems justifiable and desirable to review this observation separately at this time.

REPORT OF A CASE

The patient had been regarded as a normal healthy female infant. Nevertheless, during the course of a respiratory infection at the age of eight months, her attending pediatrician had noted some unusual findings. He had observed that the heart sounds were misplaced to the right, and a suspicion of dextrocardia had been entertained. Later he noticed what was thought to be consolidation over the right chest. A roentgenogram of the chest revealed the presence of the left lung, but on the right side of the chest there was a shadow, diagnosed as a possible fibrosis obliterans of the right lung.

When the child was two years old, she was toddling about the room one evening while eating some peanuts and chewing gum. She stumbled and fell, aspirating some partially masticated peanuts and the chewing gum into the tracheobronchial tree. She became dyspneic and cyanotic immediately. She was rushed to the hospital in a police ambulance but died in the admitting room before she could be examined. Death occurred about twenty minutes after aspiration of the foreign bodies.

At autopsy, the left lung was found to be hypertrophied; it filled the left side and extended beyond the midline. A large thymus gland occupied the upper third of the right thoracic cavity. The heart occupied the lower two-thirds. The right lung was completely absent, with the bronchus ending blindly. There was no rudimentary

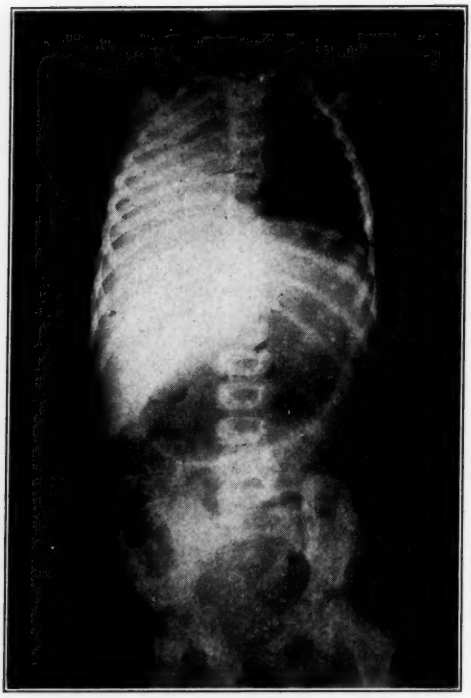

Fig. 1.—Congenital absence of right lung. X-ray of chest.

right lung tissue nor vessels. Section through the stump of the right bronchus showed nothing but cartilage. When the left bronchus was opened, two foreign bodies, consisting of masticated masses of chewing gum and peanuts, were found.

DISCUSSION

So far as could be determined by a rather careful review of the recent literature, there is only one other instance reported[2] of agenesis of one lung in which death followed aspiration of a foreign body. This patient was a boy, aged 22 months, and the foreign body was a peanut aspirated into the trachea.

Since the child I observed had survived early infancy in practically normal health, it seems likely that she belonged to the group

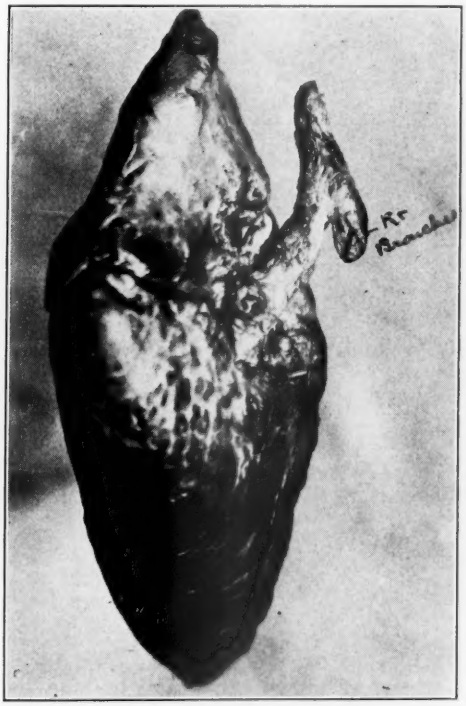

Fig. 2.—Congenital absence of right lung showing right bronchus ending blindly. Left lung much larger than normal.

of cases of agenesis of one lung in which the prognosis is relatively favorable for longevity. Barring an accident or severe disease involving the one lung present, patients in this group apparently are not handicapped nor is their general health impaired by this anomaly. There is another group of patients born with only one lung, however, who do not survive the first few days or weeks or months of infancy because of impaired respiratory function. In these cases, the picture is often complicated by the presence of other congenital anomalies.

Agenesis of one lung is of interest because of its rarity. Reinhoff[3] found only two instances in the records of Johns Hopkins Hospital which included over 150,000 cases and an additional 14,000 autopsies.

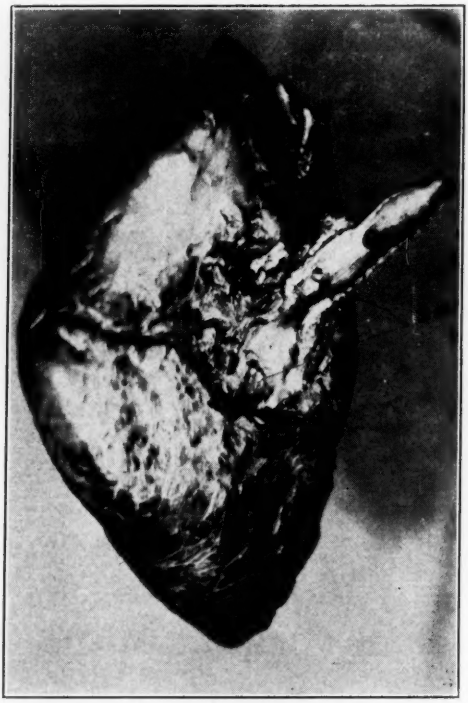

Fig. 3.—Congenital absence of right lung. Two foreign bodies in the main stem bronchus.

Olcott and Dooley[4] reported the first case observed in 10,000 autopsies, including many on newborn infants, at New York Hospital.

In 1939, Killingsworth and Hibbs[5] collected 37 cases from the literature and added one of their own, making 38. The present case was included in this group. The previous year (1938), Bohnholtzer[6] presented a review of articles in the foreign literature comprising 25 cases, of which 15 were not included in the series collected by Killingsworth and Hibbs. These additional cases listed by Bohnholtzer were by Wollmann (1891), v. Eicken (1904), Bönninger (1927), Bönninger (1931), Klinz (1787), Haberlein (1812), Gruber (1870), Gruber (1884), Lochow (1929), Clemens Wehr (1934), Anneleise Knott (1934), Riviere (1779), Pozze (1812) and Söm-

mering (1812). Bohnholtzer also added another case she had observed personally. Thus the total number of cases in these two latest reviews is 54. The two cases reported by Reinhoff[3] in 1937 were not included in either of these reviews, probably because they were reported with a series of cases of surgical ablation of one lung and were not found under this title.

Additional cases available for study since Killingsworth and Hibb's review include one reported by Jamuni and Ellis,[7] two by Wasmuht,[8] and one each by Formijne,[9] Haemstra,[10] Choisser and Bloedorn,[11] Stokes and Brown,[12] Madigan,[13] Van Loon and Diamond[14] (they also cite a case of Münchmeyer's observed in 1885, which was not cited in the other reviews), Gartside,[15] Olcott and Dooley[4] (they also allude to two cases in homozygous female twins observed by Finkelstein, apparently not included in other collective series) DeWeese and Howard[16] (they cite two recent cases reported by Castellanos and Pereiras) and Garber.[17]

This list brings the number of cases reviewed or cited by other authors to 74. The Index Medicus yielded other titles of articles in foreign journals published during the war years which were not available for review. Therefore it seems probable that a total of more than 80, possibly nearly 100, cases of agenesis of one lung have been reported in the literature. At least one case of absence of both lungs has been observed.[18]

In the cases listed by Killingsworth and Hibbs, the age distribution was as follows: Twenty-five of those they found in the literature and also their own case were in children under 12 years of age. Of these, there were three stillborn infants; five infants under one week, 13 under six months; two under three years; one, eight years; one, twelve years. Twelve adults, aged 19 to 72 years, were included in this series. In 33 cases in which sex was recorded, 20 patients were males and 13 were females. In additional cases I have collected from the literature, 23 patients were under age 12. Of these, there were three premature or stillborn infants (Gruber[6]—two cases, Bohnholtzer[6]); eight under six months (Reinhoff,[3] Olcott and Dooley,[4] Lochow[6], DeWeese and Howard,[16] Knott,[6] Wasmuht[8]—two cases, Haemstra[10]). Patients under three years were reported by Oberwarth[6] and Münchmeyer.[14] Van Loon and Diamond's[14] patient was aged three and one-half years and Gartside's,[15] seven. The two patients reported by Castellanos and Pereiras[16] were both aged ten. The girl observed by Stokes and Brown[12] was twelve. The ages of Sömmering's[6] and Garber's[17] patients were not given, but both were chil-

dren. Ages of adults in these additional cases ranged from 19 to 50, and were reported by Formijne,[9] Wollmann,[6] Bönninger,[6] Klinz,[6] Haberlein,[6] Madigan,[13] Choisser and Bloedorn[11] and Reinhoff.[3]

Olcott and Dooley,[4] Haemstra[10] and DeWeese and Howard[16] reported that the diagnosis of agenesis was made in their cases by roentgenographic and bronchoscopic examinations, and later confirmed by autopsy. Van Loon and Diamond[14] stated that Münchmeyer, in 1885, made the clinical diagnosis of absence of the lung in a boy, aged two, which was later confirmed by necropsy. In some recently reported cases, the diagnosis of pulmonary agenesis has been made only by roentgenographic and bronchoscopic examinations in living patients. Cases of this type have been reported by Stokes and Brown,[12] Madigan,[13] Van Loon and Diamond,[14] Gartside,[15] Castellanos and Pereiras[16] and Garber.[17]

These reports show that it is possible to make a diagnosis of agenesis of the lung during life, provided attention is directed toward it. As is the case with other rare conditions, it may be that this anomaly exists more often than it is diagnosed and that more cases would be recognized provided the possibility were kept in mind. Paralysis of the diaphragm on one side and diaphragmatic hernia may present similar clinical and roentgenologic findings,[12] but roentgen tomography, bronchoscopy and roentgenologic study following introduction of lipiodol into the tracheobronchial tree should yield sufficient information to make a correct diagnosis. Bronchoscopic findings may vary considerably. In some cases there is a bronchus or rudimentary bronchus on the side on which the lung is absent, and in others there is no such rudiment. If a bronchus is present, as a rule no opening can be visualized.

Bronchoscopy should be performed in every case in which this anomaly is suspected. It is the most important feature of a complete examination in confirming the diagnosis of agenesis of the lung in patients in which this condition is suspected during life, and has a real, practical significance in certain cases. For example, in the case reported by Madigan,[13] the patient was a woman, aged 23, in whom tuberculosis was suspected. She had pain in the vicinity of the inferior angle of the left scapula, a nonproductive cough, and slight dyspnea on exertion. The diagnosis was suggested by the roentgenogram of the chest, and bronchoscopy showed that the trachea was displaced to the left with no inflammatory changes in the mucous membrane, and a normal carina. The left main bronchus was present but the lung was absent on the left side. The right lung apparently

was hypertrophied. The patient's previous health had been excellent and development of symptoms was attributed to the fact that she had been doing heavy work in a factory during the war.

The cause of agenesis of the lung is not definitely known. There is disagreement among embryologists and anatomists as to whether the defect is present in the original germ plasm, or whether it results from some disturbance that occurs early in the development of the embryo. Gruenfeld and Gray[18] state that a temporary disturbance must have manifested itself after division of the single primitive pulmonary organ into the two stem bronchi but before the stem bronchus developed its first side arms. According to some authorities this would be in an embryo between 4.0 and 4.3 mm. in length; according to others, the embryonic length would be 5 to 7 mm. In some cases, agenesis of the lung exists without other structural anomalies; and in others, there are multiple anomalies. In connection with reported opinion regarding the stage of development at which the disturbance resulting in this anomaly occurs, the observation by Finkelstein[4] of occurrence of agenesis of one lung in each of two female homozyous twins is interesting. These infants lived only one week, and autopsy showed that one had a rudimentary left lung and an enlarged right lung; the other had a rudimentary right lung and an enlarged left lung.

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LVII

PENICILLIN THERAPY IN THE TRACHEA AND BRONCHI

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The value of penicillin as an adjunct to the treatment of bronchopulmonary suppuration is well accepted. The purpose of this paper is to present a critical review of the topical use of penicillin in the tracheobronchial tree, and to present information which will contribute to the evaluation of this agent, to aid in solving some of the many still unanswered questions in regard to this method of administration.

It is to be emphasized that penicillin is an adjunct rather than a substitute for the proven principles of the treatment of suppuration. This particularly applies to those conditions requiring drainage either through the air passageways or by means of surgery. The modification of the suppurative process which may be produced by penicillin must not be allowed to lessen the diagnostic study required in all cases in which bronchogenic carcinoma is a possibility.

Penicillin may be applied to the tracheobronchial tree by several different means, the more important ones being by instillation through the laryngeal syringe, the catheter, the bronchoscope or the tracheotomy tube, by atomization or by the breathing of an aerosolized solution. Of these methods, the inhalation of aerosolized penicillin has the widest application and forms the major part of this paper.

It has been shown by many workers that drugs suspended in a gas are carried into the smallest bronchi and alveoli when the particles are of small size. This method of administration has been extensively used with certain vasoconstrictor and bronchodilator drugs,[1, 2] and its therapeutic effectiveness is well known. It is essential that the particle size be of the order of one to two micra or smaller. Particles of larger size are deposited high in the tracheobronchial tree and above. Nebulizers or inhalers which produce aerosols require considerable pressure and volume of air to accomplish this, and a gas source is

usually advantageous. Oxygen is the most readily available gas for this purpose and does not alter the properties of penicillin.[3]

The simplest apparatus for the administration of penicillin aerosol consists of the nebulizer containing the penicillin solution, a cylinder of oxygen with its pressure-reducing valve and flow gauge, and rubber tubing to connect the two. In order to conserve penicillin a "Y" tube is inserted in the oxygen tubing so that the oxygen escapes to the outside except when the open arm of the "Y" is closed by the finger. This permits aerosolization of the penicillin only during inspiration.

Various modifications of this basic apparatus have been made. Barach[4] has added a glass chamber to the nebulizer in order to allow rebreathing of the exhaled penicillin. Other workers have incorporated a rebreathing bag between the patient and the nebulizer for the same reason. Hagens[5] has used an oxygen mask attached to the nebulizer. Barach[6] has added humidification of the aerosol by placing warm water in the rebreathing bag and immersing the bag in a container of hot water. This reduces the evaporation of water from the penicillin solution and the resulting increase in the concentration of the penicillin which would otherwise occur during the course of the aerosolization.

The DeVilbiss No. 40 and the Vaponefrin inhalers produce particles of the appropriate size. For the treatment of disease relatively high in the trachea or in the larynx it might be advantageous to use larger particle size; however, no conclusion is possible on this at the present time.

A flow of oxygen of four to eight liters per minute is generally used. The flow should be adjusted so that 10 to 15 minutes are required to aerosolize one cubic centimeter of the solution. The nebulizer is placed in the mouth between the teeth. If the Vaponefrin inhaler is used, the patient may close the lips and breathe through it. This is essential if a rebreathing bag or chamber is used. Following the inhalation the mouth and throat are rinsed with water. Increased absorption of the penicillin may be achieved by the use of special breathing patterns. Bryson[3] has reported 60 per cent recovery in the urine of the penicillin administered when inspiration was prolonged to 15 seconds and the breath held for 15 seconds before expiration and the expiration followed by a short rest period. Barach[6] has reported blood levels of 0.2 units per cc. following the inhalation of 50,000 units when the "Y" tube was closed at the beginning of each inspiration and opened when the inspiration was

one-half to two-thirds completed. This method included the humidification previously mentioned and the inhalation of 0.5 cc. of salt solution added as a rinse to the nebulizer.

The method used by the authors consisted of the Vaponefrin inhaler with a "Y" tube valve.

A survey of the literature reveals great variation in dosage as given by various writers, which is in contradistinction to the quite uniformly good responses reported. The smallest dosages were reported by Vermilye[7] and Hanks,[8] who administered 1,000 units dissolved in 2 cc. of solvent once a day. The highest concentration was that given by Barach[4] and consisted of 100,000 units in 1 cc. of physiological salt solution. The most frequent individual dosage reported was between 20,000 and 50,000 units dissolved in 1 cc. of salt solution. The authors have usually given 20,000 or 25,000 units.

The frequency of administration as reported in the literature was also quite varied. As previously mentioned, one treatment a day and up to 16 treatments a day have been reported, the latter by Hagens et al.[5] The patients herein reported usually started the therapy at an inhalation every three hours and after two or three days have omitted the treatments during the night.

The daily dosage of penicillin as reported in the literature has been quite varied. Bobrowitz[9] considered 50,000 units to be too small and 100,000 units to be adequate for the treatment of bronchiectasis. Segal,[2] Barach,[6] Hagens,[5] and the authors have used about 200,000 units a day and reduced the dosage after a few days to a week. As mentioned above, Vermilye and Hanks used 500 and 1000 units a day.

Physiological salt solution is the most frequently reported solvent used. Hagens[5] has reported the use of equal parts of distilled water and peppermint water, the latter being used to disguise the odor. Vermilye[7] has used distilled water. Salt solution is thought to be less irritating to the tracheobronchial mucosa and less apt to cause coughing.

Both sodium and calcium penicillin have been used; Barach[4] prefers the latter because the incidence of reactions is less. The authors have used only the sodium salt. Now that crystalline penicillin is available commercially, its properties may be investigated.

The reported duration of penicillin therapy has varied. Most reports have indicated durations of one week to several months, and Barach has had patients under treatment for over a year.

The direct application of penicillin in solution to the tracheobronchial tree has been in lower concentration than when given by aerosolization. Five hundred and one thousand units per cubic centimeter of physiological salt solution has been the concentration most frequently reported. Bobrowitz et al[9] have reported instillation of 50,000 to 250,000 units per day for four to twelve days. The authors have used concentrations of 500 and 1000 units per cc. for topical application by dropping into tracheotomy tubes and by spraying through tracheostomy openings.

The incidence of reactions to aerosolized penicillin therapy in the authors' series and in several reported series amounts to about 15 per cent. However, Vermilye[7] reported two reactions in over 200 cases, and Segal, Hanks, and Bobrowitz reported no reactions in 76 cases. The following types of reactions have been reported: urticaria, sore throat, transient fever, soreness of the tongue and gums, and redness of the skin about the site of administration. In the authors' series of 40 cases, five reactions were observed and consisted of redness of the skin about the tracheotomy wound, redness of the skin of the nose, redness, swelling and edema of the pillars and pharynx, and two cases of skin eruption.

The redness of the skin is a minor reaction and does not necessarily indicate cessation of treatment. It may be prevented by the application of vaseline to the skin prior to each treatment. The rinsing of the mouth and throat with water following each treatment is important when concentrations of 50,000 units per cc. are used and is a procedure which may reduce the number of reactions when lower concentrations are used. The use of humidification is thought by Barach[6] to reduce reactions about the mouth, tongue, and throat.

RESULTS

The authors have previously summarized the reported results of the treatment by the inhalation of penicillin aerosol of 285 cases of bronchopulmonary disease associated with infection. The great majority of these patients were improved. Since then the following reports have become available: Segal[10, 13] has reported favorably on the preoperative treatment of 14 cases of bronchiectasis and 12 cases treated medically; five cases of lung abscess were reported of which

one patient died after surgery, one was given effective preoperative preparation and three were cured; 12 patients with severe infective bronchial asthma were reported and although there was disappearance of the penicillin-sensitive organisms from the sputum, the clinical improvement was not impressive.

Bobrowitz[9] has reported good results in the treatment of 12 cases of severe bronchiectasis, as indicated by reduction in the amount, odor, and purulence of the sputum, disappearance of most organisms from the sputum, and symptomatic improvement during the treatment, but this improvement did not persist long after the treatment was stopped.

Hanks[11] has reported on 34 patients receiving penicillin aerosol therapy with acute bronchitis, bronchiectasis, chronic bronchitis, subacute bronchitis, and acute tonsillitis and laryngitis, of which two failed to respond, three showed slight improvement, six showed moderate improvement, and 23 showed disappearance of findings and symptoms.

The authors have used the topical application of penicillin in the treatment of 40 patients and the report follows.

Penicillin applied topically has been used postoperatively in seven tracheotomies and ten laryngectomies. Two to four drops of 500 or 1000 units per cc. of salt solution were dropped into the tracheotomy tubes every hour for two days and then gradually stopped. Three patients with acute laryngotracheobronchitis were so treated in addition to the administration of penicillin parenterally. Two of these patients appeared to have definitely less secretion than usual and required less aspiration. The third patient did not appear to derive any benefit from the medication. Four tracheotomized patients and the ten laryngectomized patients appeared to be benefitted by the topical application. Routine instillation of salt solution and sulfonamide and penicillin therapy in the acute laryngotracheobronchitis and laryngectomy patients were used routinely prior to and in addition to the topical use of penicillin, so it is thought that topical penicillin is a valuable adjunct to these methods of treatment.

Penicillin has been applied to six laryngectomized patients with crusting and ulceration of the upper trachea about the tracheostomy opening appearing one to six months after operation. One hundred thousand units of sodium penicillin were dissolved in 10 cc. of physiological salt solution and used over a period of one week. The so-

lution was dropped into the opening or sprayed in with an atomizer. A favorable response was observed in all after one week and treatment was continued for two weeks in all except one patient. This patient had had considerable trouble over a period of a month and salt solution and lipidol had been used with but slight improvement. This patient has continued treatment for six weeks and now shows less crusting and disappearance of the ulceration. Two tracheotomized patients with similar crusting and ulceration of the trachea were treated. One patient showed reddening of the skin about the tracheal fistula after three days and the treatment was discontinued, although he showed definite improvement. The other patient showed improvement and treatment was discontinued after two weeks.

Fifteen patients with bronchopulmonary suppuration were treated by inhalation of penicillin aerosol with the following results: four cases of proven bronchiectasis were improved, one was unimproved and remained so after a course of penicillin administered parenterally and one case complicated by pulmonary emphysema was unimproved after one week of therapy; two patients with probable bronchiectasis were improved, one of whom has continued inhalations twice a day for five months; one patient with a chronic bronchitis and pulmonary infiltration developed a sore throat with redness, swelling and edema, necessitating discontinuance of treatment without clinical evidence of improvement; three cases of suppurative bronchitis were improved; one patient with a laryngotracheitis, following partial removal of a thyroid carcinoma, during x-ray therapy developed a skin reaction to the penicillin and no evidence of improvement was noted; one patient with recurrent subacute bronchitis following upper respiratory infections was treated twice with apparent cure, in spite of redness of the skin about the nose; and one patient with large abscesses in the right upper and the left lower lobes was treated without benefit, developed a skin reaction and subsequent thoracotomy and biopsy revealed an unclassified carcinoma in the left lower lobe.

Table 1 shows the results of bacteriological studies made on 14 patients with various types of bronchopulmonary suppuration. The number of organisms present at the pre-penicillin examination was reduced in number or absent in all instances except three. One patient who was improved by treatment was reported as showing the same frequency of fusiform bacilli in smears. One patient who was not improved showed a greater growth of N. catarrhalis and Streptococcus viridans in the cultures. One patient whose smears showed

TABLE 1.—SUMMARY OF BACTERIOLOGICAL EXAMINATION OF 14 PATIENTS WITH BRONCHO-PULMONARY SUPPURATION OF WHICH 11 WERE CLINICALLY IMPROVED AND 3 WERE CLINICALLY UNIMPROVED.

	ORGANISMS PRESENT BEFORE THERAPY		ORGANISMS PRESENT AFTER AND NOT BEFORE THERAPY	
	IMPROVED	UNIMPROVED	IMPROVED	UNIMPROVED
Gram + Cocci	11	3	--	--
Gram — Cocci	2	1	3	--
Gram — Bacilli	5	--	--	--
Spirilla	2	--	--	2
Fusiform	5	2	--	1
Gram + Bacilli	1	--	--	1
Strep. Viridans	5	2	--	--
Strep. Hemolyticus	1	--	--	--
Gamma Strep.	--	--	1	--
Staph. Aureus	3	--	--	--
Staph. Hemolyticus	1	1	--	--
D. Pneumoniae	2	--	--	--
H. Influenzae	--	--	1	--
N. Catarrhalis	6	2	--	--
B. Lacto Aerogenes	--	--	3	--
Yeast	--	--	2	1
Diphtheroids	--	--	1	--
B. Coli Communis	--	--	2	--
Micrococci	1	--	--	--

fusiform bacilli received iron cacodylate intravenously and showed a reduction in the number of these organisms and clinical improvement. Inspection of Table 1 reveals that the presence of supposedly penicillin-sensitive organisms does not indicate of itself a favorable response. The appearance of spirilliform and fusiform organisms during treatment would seem to indicate that these organisms are not penicillin-sensitive and, therefore, that penicillin should not be used as a substitute for the usual chemotherapeutic agents employed in this type of infection.[14] Penicillin-sensitivity tests were not made on these patients; however, there was a predominance of the gram-positive organisms which are usually sensitive to penicillin.

DISCUSSION

Penicillin is particularly suited for topical application because it is readily soluble in water, physiological salt solution, and body fluids; it does not readily diffuse, and its potency is not affected by organic detritus.[15] McMahon[16] has questioned the effectiveness of topical application of penicillin on the basis that the infection is submucosal and that the penicillin does not reach that area. That penicillin is absorbed following topical application to the tracheobronchial tree by instillation and inhalation is indicated by the concentration in the blood and urine which follows. The absorption may, however, occur principally in the alveoli rather than through the mucosa where the infection in many of the cases is localized. It is well known that epinephrin may be topically applied by the inhalation of the 1:100 aerosolized solution, and that it is effective in the relief of dyspnea in many cases of bronchial asthma. The effect of this means of application may differ from that of parenteral administration in that a favorable therapeutic response is obtained in some cases which do not respond to subcutaneous administration. This difference in response may be a matter of concentration in the bronchial walls. White[17] and Bobrowitz[9] have found the concentration of penicillin in the sputum to be undemonstrable following the intramuscular administration; Bobrowitz has found average sputum concentrations of 86 units per cc. during the administration of 100,000 units a day by inhalation of the aerosol.

Penicillin is bacteriostatic in the ordinary therapeutic concentrations and in high concentrations it has some bactericidal properties. It may be that the relatively high concentrations in the sputum result in these bactericidal properties being active. Bobrowitz has also demonstrated penicillin in the sputum for as long as ten days after the penicillin was stopped and for eight days after it disap-

peared from the urine following ten daily intratracheal instillations of 100,000 units. The authors have had one patient who complained of the odor of penicillin for two days after the aerosolization was stopped. This suggests that topical application results in a more constant and longer exposure to penicillin than occurs following the intramuscular administration.

Proetz[18] has demonstrated on excised rabbit trachea that penicillin has little if any effect on ciliary activity in concentrations up to 250 units per cc. of isotonic salt solution; 500 units per cc. reduce ciliary activity. This susceptibility of ciliary activity to penicillin should be considered. However, the distribution of the penicillin aerosol throughout the tracheobronchial tree would probably cause dilution to below the level which affects ciliary action. The instillation of penicillin solutions in the absence of large quantities of secretion may be an indication for the use of solutions less concentrated than the 500 and 1000 units per cc., as has been used apparently without detrimental effect.

Jackson[19] and others have stressed the irritative role of the bacteriological, putrefactive changes which occur in retained bronchial secretion, particularly in bronchiectasis. It may be that topically applied penicillin diffuses through these secretions and prevents these changes, as is evidenced by the rapid disappearance of the foul odor, and causes improvement by the reduction of this source of irritation.

Two factors are important in the blood levels which result from the inhalation of a given amount of penicillin aerosol. The first is closely related to the technical means of conservation of penicillin. These are refinements in technique and consist of the thumb "Y" valve in the oxygen tubing, the rebreathing bag or container, humidification and closure of the valve only during the first half or two-thirds of inspiration. The second factor is the breathing pattern. When a slow inspiration followed by a pause and a short rest period between breaths is done, the resulting blood levels are raised over those resulting from undirected breathing. Bryson[3] has reported excretion in the urine of 60 per cent of the penicillin administered by the use of this pattern. This is nearly the rate which is observed following intramuscular administration. The significance of blood levels in terms of the effectiveness of aerosol therapy is not well understood. A high blood level would seem to indicate good penetration into the finer ramifications of the bronchi and into the alveoli. However, in the cases of bronchiectasis this deep penetration may not be essential to effective therapy. Segal[13] has reported good results

in bronchopulmonary infections in which demonstrable blood levels did not occur and concluded that blood levels are not essential for effective aerosol therapy. Other workers do not agree with this.

The reduction in the amount and the improvement in the purulence of the bronchial secretions in bronchiectasis make the inhalation of penicillin an important adjunct in the preoperative preparation of the patient for surgery. The lessening in the amount of secretion lessens the dangers of spill-over into the other lung or other lobes. The improvement in the general well-being which so frequently is seen in this type of patient is likewise of value. Olsen[20] has reported on this use of penicillin aerosol and states that the reduction in the amount of sputum was over 75 per cent.

Certain workers have stated that penicillin aerosol was more effective in acute disease than in chronic disease. When compared with the effectiveness of other nonoperative treatment of chronic bronchopulmonary suppuration, penicillin in some cases appears to be an effective method of treatment. Sufficient experience with prolonged treatment has not as yet accumulated so that an evaluation in chronic disease cannot at this time be made.

Penicillin aerosol has appeared to be more effective in some cases than penicillin administered intramuscularly. The avoidance of injection, self-medication and home treatment are aspects of penicillin therapy deserving of consideration.

Topical application of penicillin to the tracheobronchial tree either by instillation or inhalation of the aerosol is a valuable adjunct to the treatment of bronchopulmonary infection.

Aerosolization of 200,000 units in physiological salt solution for from a few days to two weeks with subsequent reduction in the dosage would seem to be adequate, although further experience may alter this.

CONCLUSIONS

The topical application of penicillin to the tracheobronchial tree and the inhalation of aerosolized penicillin are effective in the treatment of certain cases of bronchopulmonary suppuration. Although this type of application appears to be more effective in some instances than parenteral administration, further evaluation is required.

Penicillin, regardless of its mode of administration, is an adjunct in therapy, rather than a replacement for present principles of therapy.

Aerosolization of 200,000 units in physiological salt solution for a few days to a period of two weeks followed by reduction in dosage is an adequate dosage; however, more experience may alter this view.

Selection of patients on the basis of bacteriologic demonstration of susceptible organisms is valid.

The incidence of reaction to penicillin aerosol therapy may be as high as 15 per cent. Reactions constituting a serious threat to the patient have not been seen.

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LVIII

BOECK'S SARCOID: A BRIEF REVIEW AND REPORT OF A CASE IN WHICH DIAGNOSIS WAS MADE BY BRONCHOSCOPIC EXAMINATION AND BIOPSY

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ROCHESTER, MINN.

Boeck's sarcoid is a chronic systemic disease which resembles tuberculosis, leprosy and other members of the granuloma family.[10] The etiology of Boeck's sarcoid is unknown, but many authorities believe it is a form or variant of tuberculosis.[14] Boeck's sarcoid, however, is recognized as a distinct entity from both the clinical and pathologic standpoints.

The disease is as protean in its manifestations as are tuberculosis and syphilis. Today, sarcoidosis embraces a number of clinical syndromes which were originally described as separate diseases. This accounts for the variety of names which have been applied to the diverse manifestations of a single disease.

The dermatologic process was first described by Hutchinson in 1875. Besnier[2] in 1889 gave the name "lupus pernio" to the same cutaneous lesion, and Boeck[3] in 1899 was responsible for the term "sarcoid." Boeck suggested that the cutaneous lesions were parts of a systemic disease. Schaumann[15] in 1914, however, was the first to associate the cutaneous disease of Besnier and Boeck with lesions involving other organs. Schaumann coined the term "benign lymphogranuloma" to apply to the disease. In 1909 Heerfordt[6] described lesions of the uveal tract of the eye occurring in conjunction with enlargement of the parotid gland. He was responsible for the term "uveoparotid fever." In 1919 Jüngling[7] described "osteitis tuberculosa multiplex cystoides" which later was recognized as a component of sarcoidosis. At present, "Boeck's sarcoid" and "sarcoidosis" are the most common designations for this disease. It is also referred to as noncaseating tuberculosis.

The diagnosis of Boeck's sarcoid is based upon correlation of the histopathologic picture with the clinical syndrome. Thus, mi-

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croscopic examination of tissue obtained for biopsy or of specimens secured at postmortem examination has been the basis for diagnosis in most of the reported cases. Without confirmation by the pathologist, the physician alone is seldom justified in making a diagnosis of Boeck's sarcoid. The histologic unit of sarcoidosis consists of the "hard tubercle" composed of epithelioid cells, a few giant cells, a thin layer of lymphocytes and little or no inflammatory reaction in the periphery of the tubercle. Necrosis is not detected in this lesion. The process has an apparent predilection for the lymphatic system. Regardless of the site of the granulomatous lesion of sarcoid, the microscopic picture is uniformly the same.

As has already been stated, the clinical manifestations are most diverse. Nearly every structure in the body may be involved. I shall not attempt to describe the varied clinical forms of sarcoidosis as they apply to different structures. The commoner manifestations, however, should be enumerated. Particular attention will be paid to sarcoidosis of the lung.

Lesions of the skin may be nodular or may occur in plaques. They may be present almost anywhere on the body. Lesions of bones occur most commonly in the phalanges of the fingers and toes; they usually appear as cystlike areas of rarefaction. Other bones may be affected. Lesions of bones often are asymptomatic, but associated cutaneous disease is common. Mutilating changes may occur, and fusiform swellings of the fingers and toes may be noted.

The lymphatic system is almost always involved. Enlarged superficial lymph nodes often are found at some stage of the disease. Cervical, axillary, inguinal, epitrochlear, auricular and submental nodes often are demonstrated. They are seldom large and usually are not tender. Roentgenographic demonstration of mediastinal nodes may constitute the chief evidence of thoracic involvement. Bernstein and Sussman[1] stated that sarcoidosis invades the lymphatic structures of the interlobular septums and that involvement of lymphatic tissue is responsible for the characteristic roentgenographic appearance.

Sarcoidosis of the eye often is associated with enlargement of the parotid glands. This association is responsible for Heerfordt's syndrome or uveoparotid fever.[9] Sarcoidosis of the stomach[5] has been reported. Ileocecal sarcoidosis may closely resemble regional enteritis.[18] Cranial nerve palsy occurs, and usually accompanies the uveoparotid syndrome. Sarcoidosis of the heart, liver, spleen, kid-

neys, breasts, pituitary gland[4] and many other organs has been reported.

Boeck's sarcoid involving the thorax is of particular interest. Despite an alarming roentgenologic appearance, physical signs may be minimal or absent. The symptoms of which the patient complains usually are inconsequential. Bilateral enlargement of the hilar nodes is the most common observation at roentgenographic examination. Strandlike infiltrations commonly are seen radiating out from the hilus. At times a nodular appearance is seen or confluent densities are noted. Various combinations of involvement of lymph nodes and pulmonary lesions are seen. The appearance of the roentgenogram of the thorax varies greatly from time to time. The thoracic lesions of sarcoidosis may imitate a variety of pulmonary diseases, such as tuberculosis, silicosis, pulmonary fibrosis, leukemia, primary or metastatic malignant disease and especially lymphoblastoma.

The cutaneous, osseous and visceral forms of sarcoidosis may occur alone or in any combination with each other. The disease progresses slowly and the clinical picture is likely to vary from time to time. The extent of the disease process frequently is unrelated to the general appearance and well-being of the patient. Many patients who have extensive disease have no symptoms at all. Dyspnea and cough may be present if pulmonary involvement is extensive. Involvement of vital organs, such as the heart, nervous system or kidneys, may produce very serious clinical symptoms or even death.[8] On the whole, the prognosis of Boeck's sarcoid tends to be good, although some patients do die of the disease. It is remarkable that extensive disease frequently will disappear and leave little residue. A few patients succumb with extensive active tuberculosis.

With the exception of roentgenography, the tuberculin test is probably the most important of the laboratory procedures. In the presence of sarcoidosis, the reaction to the tuberculin test almost invariably is negative. The problem of tuberculin anergy and its relationship to sarcoidosis and tuberculosis is beyond the scope of this paper. When, however, histologic proof of sarcoidosis has been obtained, it is most unusual ever to encounter a positive reaction to the tuberculin test. It is felt by some authorities that sensitivity to tuberculin in a patient who has sarcoidosis may indicate that frank tuberculosis is developing.[13]

It is usually stated that the leukocyte count will be normal or low in the presence of Boeck's sarcoid. The number of monocytes and eosinophils may show an increase at the expense of the number of neutrophils. The sedimentation rate usually is increased. The value for serum proteins may be slightly higher than normal, and there may be a relative increase in the serum globulin.[17] Examination of the sputum for Mycobacterium tuberculosis invariably is negative. Material aspirated from the fasting stomach of a patient will not kill guinea pigs when they are inoculated with it.

The treatment of sarcoidosis has been largely empiric. Treatment is very difficult to evaluate because of the slow progression of the disease and its tendency toward gradual improvement. Arsenical compounds, iodides, tuberculin and hyperpyrexia have been used from time to time. Some authorities feel that gold salts can be used to advantage; this form of therapy was employed in the case at hand. Recently, roentgen therapy has been advocated.[12] The administration of an ester of chaulmoogra oil (chaulmestrol), especially in combination with roentgen therapy, has been used recently, and preliminary results are somewhat encouraging.

REPORT OF A CASE

A woman 47 years old registered at the Mayo Clinic on April 1, 1940. She complained of difficulty in swallowing and talking. Her symptoms had arisen rather suddenly in September 1936, when she had regurgitated fluids through the nose. At the same time, she had noted huskiness of her voice. Her local physician had examined her throat and found paralysis of the right vocal cord and right side of the hypopharynx. The symptoms had abated and she had felt that she was in normal condition again after two months.

In December 1938, her physician had re-examined her throat and found that the right vocal cord was still paralyzed. She had had no complaints to make at that time.

In November 1939, dysphagia had recurred. Food and fluids were regurgitated again. Difficulty in speech had recurred. Because these symptoms were not abating, the patient had come to the clinic.

A history of several episodes of severe epigastric distress and pain in the right upper quadrant of the abdomen was elicited. This was referred to the back and was suggestive of biliary colic. Otherwise, the health of the patient was good. No other symptoms could be elicited.

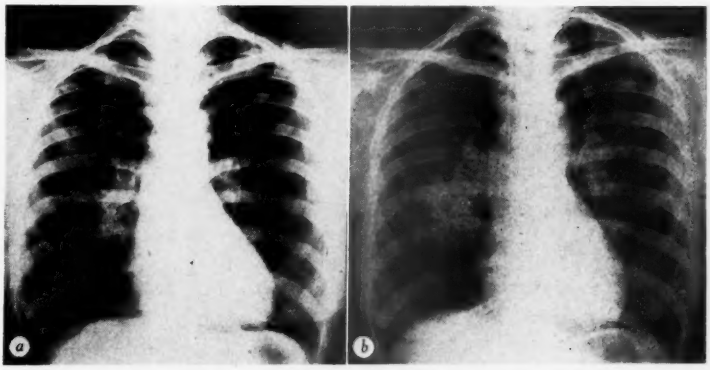

Fig. 1.—*a*, Roentgenogram of the thorax made on July 18, 1940, showing infiltrations at the second interspace anteriorly on the right, with a mass in the region of the right hilus and enlargement of left hilar shadow; *b*, roentgenogram of the thorax made on September 2, 1942, showing marked increase in size of the hilar lesions and extensive parenchymal involvement.

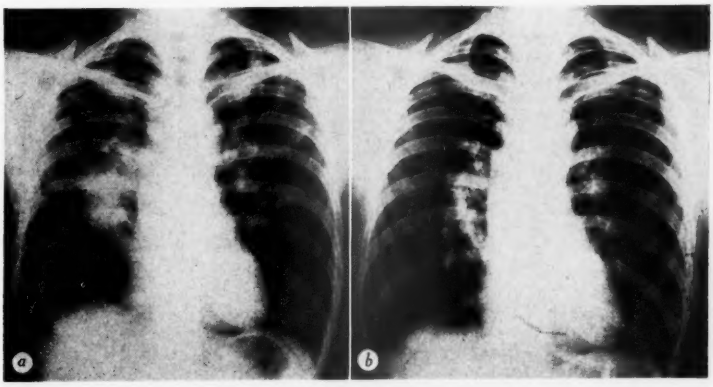

Fig. 2.—*a*, Roentgenogram of the thorax made on August 6, 1943, showing that the pulmonary lesions have become somewhat more discrete, but that extensive involvement remains; *b*, roentgenogram of the thorax made on January 18, 1945, showing marked resolution of the hilar and parenchymal infiltrations.

Examination of this patient at the clinic disclosed paralysis of the right vocal cord, of the constrictor muscles of the right side of the pharynx, and of the right side of the soft palate. Results of routine physical examination and detailed neurologic examination were otherwise negative. A diagnosis was made of palsy of the ninth and tenth cranial nerves, presumably caused by an inflammatory lesion in the brain stem.

Results of routine laboratory tests, including urinalysis, blood counts and the flocculation test for syphilis, were normal. Stereoroentgenograms of the thorax revealed an infiltration in the upper field of the right lung suggestive of pulmonary tuberculosis. Results of roentgenograms of the skull were negative. Examination of the cerebrospinal fluid gave normal results. The sedimentation rate was 31 mm. in one hour (Westergren). Although the patient had no pulmonary symptoms, a specimen of sputum was obtained. Mycobacterium tuberculosis was not demonstrated. Guinea pigs were inoculated with the contents of the fasting stomach. Tuberculosis did not develop in these animals.

In view of the possible presence of active tuberculosis, a rest regime was outlined. The patient returned to the clinic in three months (July, 1940) for re-examination. There had been no change in the patient's symptoms and the neurologic lesion had not altered. There was no significant change in the appearance of a roentgenogram of the thorax (Fig. 1*a*).

The patient again visited the clinic in September 1942, because of further attacks of pain in the upper right quadrant of the abdomen. There had been no change in the voice. She had continued to have difficulty in swallowing. A dry cough had developed. She had lost 13 pounds (5.9 kg.).

There were no additional physical observations. Cholecystograms revealed cholelithiasis. Roentgenograms of the thorax indicated marked progression of the pulmonary lesion (Fig. 1*b*). The infiltration in the right lung had become more extensive, and a lesion in the left lung appeared to extend from the left hilus. There appeared to be hilar adenopathy bilaterally. The leukocyte count was 7,600 per cubic millimeter of blood. The sedimentation rate was 50 mm. in one hour (Westergren). Results of tests with first and second-strength tuberculin (P.P.D.) were negative. The value for serum protein was 7.6 gm. per 100 cc. of serum and the A-G ratio, was 2.3:1. The value for calcium was 10.3 mg. per 100 cc. of serum and that for alkaline phosphatase was 2.3 units (Bodansky) per 100

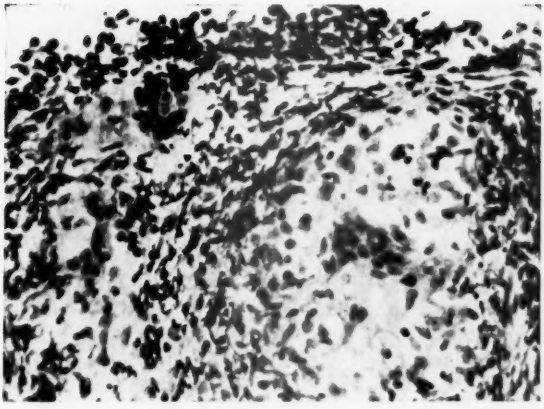

Fig. 3.—Photomicrograph of tissue removed from the lesion in the right main bronchus, consisting of the "hard tubercle" of Boeck's sarcoid (hematoxylin and eosin). x260.

cc. of serum. An electrocardiogram revealed left axis deviation with low amplitude of the T wave in lead I, and inversion of the T waves in leads II and III. Roentgenograms of the hands and feet did not reveal evidence of osseous disease, except for slight osteoporosis.

The presence of Boeck's sarcoid was suspected. A diligent search was made for palpable lymph nodes or a cutaneous lesion significant enough to warrant biopsy, to no avail. Bronchoscopic examination was carried out. The mucosa of the lateral wall of the right main bronchus had an unusual nodular appearance; a specimen was taken for biopsy. The pathologist made a diagnosis of a chronic granuloma resembling tuberculosis. The histopathologic appearance was consistent with a diagnosis of sarcoid (Fig. 3).

In the next five months the patient was treated with gold sodium thiosulfate. She returned to the clinic in February 1943. Subjectively, there had been some improvement in the voice, but the dysphagia was unchanged. Her cough was slightly improved. She was not dyspneic. Cholecystectomy was performed with the patient under the influence of spinal anesthesia. The recovery was uneventful.

The patient has not been seen at the clinic since 1943. Her local physician, however, has corresponded with us and has sent us

roentgenograms of the thorax made at regular intervals (Fig. 2*a*). The patient reported progressive abatement of all her symptoms. She said her voice is almost normal and that she has little difficulty in swallowing. Roentgenograms of the thorax have indicated progressive improvement (Fig. 2*b*). At the time she wrote, she was feeling well and was able to carry out her household duties.

SUMMARY AND COMMENT

A case of Boeck's sarcoid is presented. The history of the illness of this patient, at the time of this report, now covers a period of ten years. Lesions present involved the lungs and the ninth and tenth cranial nerves. Diagnosis of sarcoidosis was established by bronchoscopic examination and biopsy of tissue taken from the bronchial mucosa. Roentgenograms of the thorax are reproduced which demonstrate the progression and regression of the pulmonary lesions in the course of six years. Gold sodium thiosulfate was administered after a definite diagnosis had been established. It appears to have been of benefit.

To the best of my knowledge, this is the only reported case of Boeck's sarcoid in which a diagnosis was made by bronchoscopic examination and biopsy of a bronchial lesion. Bronchial and tracheal lesions of this disease have been demonstrated at necropsy,[11] and there are five reports of sarcoidosis of the larynx.[16] I have carried out bronchoscopic examination in several patients suspected of having Boeck's sarcoid, but in no other instance has a tissue diagnosis from the bronchial mucosa been obtained.

The case at hand is of particular interest because of the lesions of the cranial nerves. Involvement of cranial nerves has been reported not infrequently as accompanying sarcoidosis, but it usually occurs in association with the uveoparotid form of the disease, and the seventh nerve is most commonly affected. I believe I am justified in assuming that the lesions of cranial nerves in this case were caused by sarcoidosis. The improvement in speech and deglutition is consistent with the course in many cases of sarcoidosis.

Boeck's sarcoid is a condition which must be kept constantly in mind by those interested in thoracic disease and bronchoscopy. Although this condition is most commonly confused with pulmonary tuberculosis and Hodgkin's disease, it also must be distinguished from primary or metastatic malignant disease of the lung, leukemia, pulmonary fibrosis, bronchiectasis and fungous disease of the lung.

MAYO CLINIC.

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LIX

POINTS OF MUTUAL INTEREST IN BRONCHOLOGY AND ANESTHESIOLOGY

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AND

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Both the bronchologist and the anesthesiologist are actively interested in the study of respiration. Each deals directly with cases of respiratory disease or disturbances of respiration. In the mutual approach to these problems through the larynx, trachea and bronchi, the separate points of view enhance each other and give opportunities for greater cooperation leading to better service to the patient. This paper will consist of an analysis of the interlocking phases of the two fields and methods of their integration for mutual benefit.

During any surgical procedure, both the bronchologist and the anesthesiologist are concerned with the general condition and welfare of the patient, but in nearly all the problems involved, their viewpoints are slightly different. The bronchologist is accustomed to dealing with a patient who presumably has some pathologic condition which jeopardizes his welfare or his life. He must diagnose and, if possible, treat that condition with the ultimate prognosis uppermost in his mind. His manipulations must be carried out skillfully; and, assuming that a reasonable amount of skill is used, these manipulations do not of themselves add much hazard to the patient's welfare. Therefore during his surgical procedures the bronchologist focuses his attention on his work and on the local condition he is treating. Although always alert to signs of impending trouble he cannot easily divide his attention between the surgical procedure and the patient's general condition.

The anesthesiologist, on the other hand, although also concerned with the patient's ultimate welfare, is far more accustomed to focusing his attention on the immediate condition of the vital functions, particularly respiration and circulation. This is true for several reasons. First, the indications for surgical intervention and the probable benefit to be derived are primarily the surgeon's respon-

sibility; if the surgeon concludes that a patient requires an operation, the anesthesiologist's function is to administer the best anesthesia and to use all his ability to insure that the patient survives the operation, the anesthetic, and the postoperative difficulties. Second, the administration of an anesthetic agent puts the patient's welfare and his life in jeopardy, and the anesthesiologist is accustomed to watching zealously for the slightest indication that the patient's condition is being adversely affected. Third, as guardian of the patient's welfare during the whole procedure, the anesthesiologist is trained to anticipate the surgeon's requirements as well as the nature and degree of interference with the patient's vital functions which the operation may entail. He accepts the role of guardian and expects the surgeon to trust him to watch the patient's general condition, allowing the surgeon to give his undivided attention to the operative procedure, unless warned that the condition is unsatisfactory.

Thus we see that the points of view of the two specialists are similar, yet differ in important respects. When a bronchologist requires the assistance of an anesthesiologist, a mutual understanding of the role that each must play leads to satisfactory cooperation. The surgeon can devote his almost undivided attention to his work. The anesthesiologist must provide the best working conditions. He must know the surgeon's procedure and its requirements. He must guard the patient's general condition.

The overlapping points of view in the two specialties are well exemplified by the mutual interests regarding the respiratory mechanism. Disturbances of respiration are such characteristic complications of anesthesia that the anesthesiologist, probably more than any other specialist, becomes an expert in this field.

He sees respiratory obstruction, respiratory depression and other aberrations of the respiratory mechanism daily, almost hourly. His attention is constantly focused on the dynamic aspects of respiration, the immediate need for adequate respiratory exchange, the dangers of lack of oxygen and of carbon dioxide accumulation. He is much more concerned with the immediate dynamic aspects of respiration than he is with chronic pathologic conditions of the lungs.

The bronchologist meets many similar problems. He sees fewer cases of depression and interference with the nervous control of respiration, but he deals frequently with respiratory obstruction. The difference is that he deals chiefly with causes of obstruction which require surgical removal (foreign bodies, tumors), whereas the anesthesiologist sees more functional disturbances (pharyngeal

obstruction, laryngospasm) which require temporary relief. Some conditions, such as debris in the bronchi, are common problems confronting both specialists.

Methods of examination and treatment used in the two specialties are similar in certain respects. Laryngoscopy is a procedure common to both and to a large extent the anesthesiologist has copied the equipment and the methods of the bronchologist. The bronchologist uses his laryngoscope for examination of the pharynx and the larynx and through it he passes straight rigid instruments, such as forceps or a bronchoscope. It serves his purpose best to have a laryngoscope which is circular in transverse section with a removable slide making up its posterior surface. The anesthesiologist also uses a laryngoscope for exposure of the larynx, but through it he usually passes flexible curved rubber tubes. This technic differs from the passage of a bronchoscope. Since one cannot see through the endotracheal tube, he must try to see around it as it passes through the laryngoscope. For this reason the laryngoscope has a wide opening on the right side and in transverse section it is U-shaped. The endotracheal tube is slipped in from the right side, only the distal end occupying the lumen of the laryngoscope. Some endotracheal tubes, however, are constructed of metal, are rigid and straight when inserted and closely resemble a bronchoscope.

One method of intubation which the anesthesiologist frequently uses is entirely foreign to the bronchoscopist, viz., that of blind intubation. The tube is passed through the nose into the pharynx and then manipulated until the tip enters and passes through the larynx. This is of particular value in providing a free airway in cases in which direct laryngoscopy is difficult or impossible.

In both bronchoscopy and anesthesia local anesthetics are commonly used. The bronchologist does the majority of his procedures under topical anesthesia and is familiar with the technic and hazards of this method. The anesthesiologist also uses topical anesthesia for intubation in some cases. With his experience with other types of regional anesthesia he is familiar with the hazards involved in the use of these drugs. Both specialists are prepared to recognize and to treat toxic reactions. However, with his greater familiarity with resuscitation and his frequent use of intravenous barbiturates, such as pentothal sodium, the anesthesiologist is probably better prepared and better equipped to treat these conditions. They may involve circulatory collapse requiring the administration of oxygen, vasoconstrictors, and perhaps artificial respiration; or they may in-

volve convulsions requiring the administration of oxygen and an intravenous barbiturate, and possibly artificial respiration. When such toxic reactions occur, it may be advantageous to call for the assistance of an anesthesiologist if one is available, but the bronchologist must be capable of dealing with them himself. Such assistance is invaluable since the efficient airway obtained with a rubber endotracheal tube and the regular respirations continued over a long period of time with the oxygen bag maintain life while the toxic reaction is being controlled with suitable measures.

Although many of the conditions for which bronchoscopy is indicated are beyond the sphere of the anesthesiologist, one condition in which both specialists have an equal interest is atelectasis. This condition is frequently caused by the plugging of a bronchus by mucus or other debris with consequent absorption of the atmosphere in the distal alveoli and collapse of the affected part of the lung. The condition develops not uncommonly during anesthesia or in the postoperative period. If it is not relieved early by conservative measures, it becomes more and more important that the offending plug be removed by suction. The bronchologist is frequently called on to carry out this treatment. The anesthesiologist is accustomed to attempting the same thing by inserting an endotracheal tube and then passing a suction catheter into the bronchi through this tube. Admittedly this is a less specific treatment than bronchoscopic suction. There are many similar occasions at the end of anesthesia when the tracheobronchial tree may be grossly contaminated and, although atelectasis may not be developing, the debris should be removed. At this point there is opportunity for the greatest cooperation between the bronchologist and the anesthesiologist. At the completion of the surgical procedure the trachea and both bronchi are cleared of debris by the anesthesiologist by means of the suction catheter passed through the endotracheal tube. The pharynx is then thoroughly cleaned and the catheter and the endotracheal tube removed. A bronchoscope is immediately inserted and the remaining secretions rapidly removed by bronchoscopic aspirators passed into the individual branch bronchi under direct vision. As the bronchoscope is withdrawn, the responsibility for the patient's airway is returned to the anesthesiologist. This moment is, at times, a very critical one and the advantages of cooperation must not be confused with the very great disadvantage of divided responsibility. This is so easily the case that an absolute understanding must be pre-arranged.

The conception that suction bronchoscopy should legitimately be regarded as coming within the anesthesiologist's field has been

growing. A capable bronchoscopist is not always available at a time when this treatment is indicated, whereas suction with a catheter is not always adequate. With his knowledge of laryngoscopy and his familiarity with the tracheobronchial tree as a background, the anesthesiologist is quite capable of being instructed in the technic of bronchoscopic aspiration. Certainly no one should attempt bronchoscopy without adequate preliminary instruction. Similarly the anesthesiologist must strictly limit his use of the bronchoscope to the removal of debris by aspiration. Bronchoscopic diagnosis is definitely not his field, and he must not attempt the removal of foreign bodies except under dire circumstances when no bronchologist is available and the time will not permit calling one.

If it is definitely understood that injudicious interference with foreign bodies may do more harm than good and that ability to insert a bronchoscope does not make a diagnostician, it would seem that the anesthesiologist might justifiably encroach to this limited extent on the field of bronchoscopy.

If it is appropriate that the anesthesiologist should appeal to the bronchologist for training in one aspect of that specialty, it would be equally appropriate that the bronchologist should appeal for training in some aspects of anesthesiology. Experience gained in the administration of general anesthesia leads to better understanding of the dynamics of respiration, the transport of blood gases, the recognition of respiratory emergencies, and the use of resuscitative and sedative measures. A period of from one to three months spent in a good anesthesia department would be valuable training for the future bronchologist.

Although many of the surgical procedures of the bronchologist are carried out under topical anesthesia, there are certain cases in which general anesthesia is indicated. Some of the operations involve painful manipulations which cannot be prevented by topical anesthesia. Some patients are so nervous and uncooperative that even if the procedure is painless, the surgeon may not have adequate working conditions. This is especially true of some children. There are other cases in which due to anatomic peculiarities or tenseness of the patient, adequate relaxation in the conscious patient cannot be obtained. In all such cases, the use of general anesthesia should be considered.

Certain specific methods for dealing with some of the problems which may arise may be mentioned. For laryngoscopy and bronchoscopy it has been found that ether anesthesia usually provides the

most satisfactory relaxation and obtundation of reflexes. Greatest difficulty in the use of ether in these procedures arises from the fact that the surgical intervention interferes with satisfactory continuation of the administration of the anesthetic by inhalation. During laryngoscopy, a certain amount of etherization may be maintained by insufflation. During bronchoscopy this is not convenient or effective because the flow of ether vapor down the side tube of the bronchoscope results in refractive disturbances which interfere with the bronchoscopist's clear vision. For prolonged procedures rectal ether anesthesia has been found to give excellent working conditions. If the ether given by rectum does not produce an adequate depth of anesthesia, further etherization may be accomplished by administering ether by inhalation. Once a satisfactory level of anesthesia has been established, it will be maintained without further administration for a long time. There is very little reflex response and sometimes the surgeon's only complaint has been that the larynx was so relaxed that recognition of the true phonating edges of the cords was difficult. The greatest difficulty with rectal ether lies in the fact that the effect lasts for a number of hours and constant postoperative care may be required for five or six hours following the operation.

Another agent which may be administered by rectum is tribromethanol (avertin). This is particularly valuable in cases where a cautery or other source of ignition of inflammable anesthetic is to be used. However, the dosage of the drug has to be higher than we ordinarily desire. Also there is again a prolonged postoperative depression.

It would seem that an ideal solution to the problem would be the use of a short-acting intravenous anesthetic such as pentothal. However, it has been found that this is an unsafe procedure. Respiratory reflexes are not easily abolished by this drug and even with the additional use of topical anesthesia, hazardous laryngospasm and expiratory spasms (prolonged sustained coughing) are apt to result in serious lack of oxygen.

For esophagoscopy the situation is much simpler. An endotracheal tube may be passed either through the mouth or through the nose and anesthesia maintained through this. The anesthetist then has constant control of the depth of anesthesia. The tube does not interfere with the insertion of the esophagoscope and it does provide a free airway preventing the occurrence of laryngospasm.

For many cases of tracheotomy it has been found that an endotracheal tube provides for the maintenance of the airway and thus facilitates the surgical procedure. In some cases where a severe obstruction has been present, when the patient arrives in the operating room a bronchoscope has been inserted. In extremely apprehensive or uncooperative adults an endotracheal tube may be inserted under topical anesthesia and a general anesthetic safely administered. This avoids severe obstruction which may develop during induction. For patients who are unconscious because of oxygen want, no topical anesthesia is necessary. Patients with edema of the larynx, such as may develop from an abscess in the neck, are best anesthetized by the endotracheal technic. In one case complete respiratory obstruction due to edema developed while the patient was waiting in the operating room. The prompt insertion of the endotracheal tube prevented a fatal outcome, or the necessity for a tracheotomy. The abscess was drained and the tube was removed several hours later when the edema had subsided.

For external laryngeal surgery the use of an endotracheal tube is nearly always an asset. The technic for laryngectomy requires some modifications. The tube is inserted either orally or nasally in the beginning of the procedure. After the surgeon has freed the larynx and is ready to sever the trachea, the tube is removed, the trachea opened, and an Anode endotracheal tube which is very flexible is inserted by the surgeon through the tracheostomy opening and the other end of the tube passed up under the drapes to the anesthetist for maintenance of inhalation anesthesia. An inflatable cuff on the end of the tube prevents blood from trickling down the trachea; the anesthetist removes secretions through this tube with a small suction catheter and secretions are not coughed up into the operative field.

CONCLUSIONS

It is evident from all the above considerations that the anesthesiologist has a great deal to contribute to the work of the bronchologist and it is to be hoped that bronchologists will increasingly recognize the importance of having the cooperation of the anesthesiologist in many cases where his assistance is of value. In cases of toxic reactions to local anesthetic drugs, the presence of an anesthesiologist may prove of extreme value.

Surgeons who have been accustomed to seeing anesthetics administered by technicians may have had no opportunity to appreciate the scope of the anesthesiologist. The administration of anesthetics

is not merely a technical procedure. It involves great hazards for the patient and requires judgment as to choice of agents and methods, dosage, diagnosis of complications, and the institution of appropriate treatment. The patient's life and welfare are placed in jeopardy whenever an anesthetic is administered. It has been the zealous policy of the medical profession to restrict to itself the prerogative of jeopardizing a patient's welfare in the hope that benefit would result. It seems deplorable that in the one field of anesthesiology this prerogative has been delegated to lay technicians, who are quite incapable of using the fine judgments required. For this reason as well as to obtain the benefit of a wide selection of procedures, the bronchologist as well as other surgeons are benefited by the assistance of trained anesthesiologists.

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LX

DIAGNOSIS OF BRONCHOGENIC CARCINOMA BY EXAMINATION OF BRONCHIAL SECRETIONS

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AND

PETER A. HERBUT, M.D. (by invitation)

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In recent years remarkable progress has been made in the diagnosis and treatment of bronchial carcinoma. Roentgenography and bronchoscopy are now almost universally available and with advances in medical and surgical therapy the diagnosis of carcinoma should be made earlier and the mortality rate should decrease. A review of the literature indicates that these advances are recent. In 1926, while discussing bronchial carcinoma, McCrae[1] pointed out that, since a great majority of primary carcinomas of the lung have their origin in a bronchus, bronchoscopy with biopsy can give information which is obtainable in no other way. He urged bronchoscopy as a diagnostic aid and stressed the importance of early diagnosis. During the same year, Grove and Kramer[2] reported on a series of 24 cases of primary carcinoma of the lung. Twenty-one of these were diagnosed at autopsy and three by biopsy of metastatic lymph nodes. In none had there been a bronchoscopic examination. They concluded that "with more frequent use of bronchoscopy and with an appreciation that primary carcinoma of the lung is not rare this clinical entity will be diagnosed more commonly antemortem. The use of the bronchoscope both for diagnosis and treatment offers a field in a condition where formerly the prognosis was very poor."

Two decades have passed since these observations were made. Medical literature bears witness that hundreds of cases of carcinoma of the bronchus have been diagnosed during life by bronchoscopy and biopsy. Thoracic surgeons have met the challenge and in 1933 the first successful pneumonectomy for carcinoma was performed by Graham. In spite of this, however, there still remains a remark-

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able disproportion between the large number of reported cases of primary carcinoma and the small number that have been successfully treated.

Irrespective of the outstanding progress that has been made all evidence seems to indicate that the early diagnosis of bronchial carcinoma is accomplished in a minority rather than in a majority of cases, this in spite of the fact that the reported series of cases show that a high percentage of positive diagnoses has been made antemortem. A study of this indicates that there are several reasons for delay in diagnosis. One of these is failure of the patient to consult his physician during the incipient stages of the disease. The common reason is that during this stage there are few or no symptoms. This situation can be overcome only by a program of education making the patient cancer-conscious and, more important, educating the laity to have routine roentgen examinations. Mass roentgen surveys have demonstrated that not only carcinoma and other tumors but tuberculosis and many diseases are recognized long before the patient is aware of their presence and before symptoms have developed.

A second cause for failure of early diagnosis is delay in utilizing available diagnostic methods because the physician has not developed a sufficiently strong suspicion of carcinoma; in other words, he is not cancer-minded. The early clinical course of carcinoma is misleading because the symptoms either are absent, are mild or may masquerade as some other process. As a result patients are treated for bronchitis, pneumonitis, or other condition without having had a thorough examination. Overholt[3] emphasized this in his report of 153 patients with bronchial carcinoma, 60 per cent of whom were treated on the basis of an incorrect diagnosis.

As bronchologists we can accomplish little for a patient who has been under treatment many months for bronchial carcinoma erroneously diagnosed as unresolved pneumonia, pulmonary abscess, tuberculosis or other condition and finally develops atelectasis of a lobe or lung. There can be little hope for improvement until certain signs and symptoms occurring in an adult immediately suggest to his medical attendant that carcinoma must be considered as a possibility and that appropriate roentgenographic studies and bronchoscopy should be carried out. We can contribute more effectively, however, with the means at our command if we do not limit our field of usefulness only to that small segment of the bronchial tree which can be visualized through the bronchoscope. This statement

may appear gratuitous when one recalls that a large proportion of primary carcinomas occur at or near the hilum, involving the main bronchi or the primary divisions. Also, Ochsner and his associates[4] stated that bronchoscopy should give a positive diagnosis in about 70 per cent of cases of bronchial carcinoma, Holinger and his group[5] reported positive biopsies in 78 per cent of a series of 175 cases, Overholt[3] reported 62 per cent of positive bronchoscopic biopsies, and a positive bronchoscopic diagnosis was obtained in 89.3 per cent in a series of 122 cases reported by Adams.[6] In addition, a considerable number of positive diagnoses have been made bronchoscopically on the basis of deformity, fixity and stenosis of a bronchus.

In reviewing a series of 336 cases observed at the Bronchoscopic Clinic, Jefferson Hospital, from 1930 to 1945, inclusive, in which a diagnosis of carcinoma was made, it was noted that relatively few of the cases in which a positive bronchoscopic biopsy was secured were suitable for surgical treatment. It appeared that the percentage of positive bronchoscopic biopsies bears a definite direct ratio to the percentage of inoperable cases. In the large number of cases reported few authors show a rate of successfully operated cases higher than 10 per cent.

While published statistics vary, the majority indicate that approximately 50 per cent of primary carcinomas involve either the upper middle or lower lobes of the lung. Further, the cases that can be treated surgically usually are included in this group, since carcinoma of a main bronchus rarely is operable unless discovered in its incipiency. Since from 30 to 40 per cent of cases occur beyond the range of vision of the bronchologist, these offer a direct challenge diagnostically for they are the cases that more often must be diagnosed either by exploratory thoracotomy, aspiration biopsy or other means and frequently are not brought to a final conclusion diagnostically until other plans of diagnosis and therapy are tried and valuable time is lost.

Upper lobe lesions are difficult to diagnose. While a retrograde telescope may be employed, it has been of relatively little assistance. Pneumothorax is advocated but it has limitations and often does not improve visualization of an upper lobe bronchus.

Study of sputum has been advocated and while reports from England, other European countries and elsewhere appear to give a high percentage of positive findings, few cases have been recorded in North American literature. Holinger and his group[5] reported two positive sputum biopsies in 175 cases. Adams[6] reported positive

findings in but three cases. Our results have been similar to these. In our experience the examination of bronchoscopically secured secretion for cancer cells offers a method which can be employed to establish a morphologic diagnosis and is particularly valuable in lesions located in the upper pulmonary lobes and in the smaller bronchi beyond the range of bronchoscopic vision.[7]

Bronchoscopically secured secretions were selected instead of sputum for secretions are scant in early carcinoma and become profuse only when there is obstruction and suppuration in the more advanced stages of the disease. Sputum is more dilute than bronchial secretion. By bronchoscopy one can secure secretions from the region of the suspected neoplasm. Studies have shown that these cells exhibit less degenerative change and appear in greater numbers than those of dilute secretions from the trachea or sputum.

Methods of Securing Secretion.—In all suspected cases of carcinoma routine bronchoscopy is performed in the customary manner. Preliminary roentgenograms offer assistance in localizing the lesion to a particular portion of a lobe or lung. If bronchial secretions are profuse, those in the trachea and the larger bronchi can be discarded since it is desirable to secure them from the region of the lesion and to avoid dilution. When secretions are scant, all should be secured. Flexible tipped straight and curved aspirating tubes are employed. The secretion should be undiluted by sodium chloride solution because of difficulties in preparation of smears, therefore a type of collector attached directly to the aspirator is desirable. As soon as the material is obtained it is sent to the laboratory where smears are made and, while still wet, are fixed with equal parts of ether and 95 per cent alcohol. They are then stained by the Papanicolaou technic.

In the earlier cases when secretions were scant, the aspirating tube was washed with a small quantity of sodium chloride solution to obtain a specimen. Few of these were satisfactory for staining. A small gauze sponge on a carrier now is employed to secure secretions from the smaller bronchi in these cases. This is immediately smeared on slides and fixed in ether and alcohol. As alternatives, an attempt may be made to secure secretions from the tip of the aspirating tube for immediate smearing on slides or a small quantity of physiologic sodium chloride solution may be instilled into the suspected bronchus and these washings collected by aspiration. The technical problems of preparation of the smears are increased but this offers an additional means of securing secretion for study.

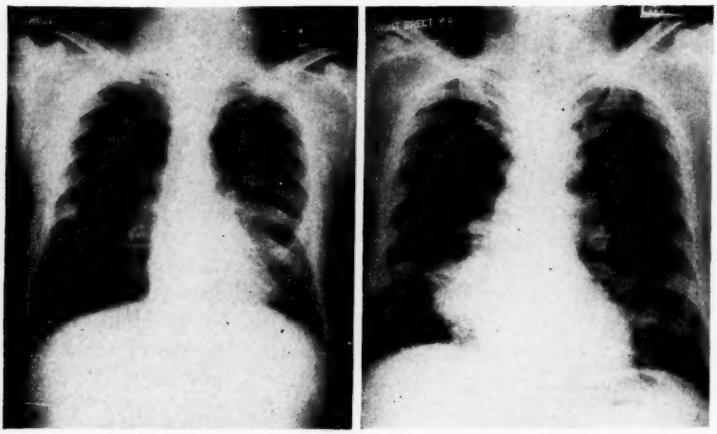

Fig. 1.—Roentgenogram revealing a shadow in the left lung occupying the lower part of the upper lobe and the perihilar region. Appearances suggested bronchogenic neoplasm. (Film by Dr. Paul C. Swenson.)

Fig. 2.—Roentgenogram showing a dense shadow extending outward from the right border of the heart, intimately connected above with the hilar shadow and occupying the upper part of the lower lobe. The appearances suggested neoplasm although an acute infectious process could not be ruled out. (Film by Dr. T. Eberhard.)

Results.—In an earlier communication there were reported 42 cases which were considered seriously as carcinoma.[7] In 38 a diagnosis of carcinoma was proved by bronchoscopic biopsy, exploratory thoracotomy, biopsy of metastatic lymph nodes, at necropsy or by death with obvious metastasis. In 30 of these, secretions were obtained bronchoscopically and in 22 cancer cells were found (73 per cent); in this same group a histologic diagnosis of carcinoma based on bronchoscopic biopsy was made in 11 cases (36.6 per cent). In an additional 7 cases a bronchoscopic diagnosis of carcinoma was made on the basis of bronchial deformity, fixity, rigidity or stenosis and in 5 of these cancer cells were obtained in secretions. There was one failure to find cells in secretions in a case proven as carcinoma by bronchoscopic biopsy and in two cases in which the diagnosis was made by observing bronchial deformity, fixity and stenosis. These were among the earlier cases and the failures were due to faulty technic in securing secretions and in staining. In spite of these fail-

ures the results of the cytologic studies were gratifying and warranted further investigation. This preliminary report indicated that the number of proved cases diagnosed by study of secretions was twice the number discovered by bronchoscopic biopsy.

During the past five months, 27 additional proved cases of carcinoma have been observed. In 25, or 92.5 per cent, a diagnosis of carcinoma was made by cytologic study of bronchoscopically secured secretion and in 13, or 48 per cent, a positive bronchoscopic biopsy was secured. In 8, or 29 per cent, there was bronchoscopic evidence of bronchial stenosis, deformity or fixity. In the entire group of 57 cases a cytologic diagnosis was made in 47 cases (82.4 per cent). A diagnosis by bronchoscopic biopsy was made in 24 cases (42.1 per cent) and by anatomico-pathological changes in 15 cases (26.3 per cent), a total of 39 bronchoscopic diagnoses (68.4 per cent). In this group of 39 cases a morphologic diagnosis by cytologic examination was made in 35. In addition a cytologic diagnosis of carcinoma was made in 12 cases which were negative on bronchoscopy.

TABLE 1.

Total proved cases	57
Diagnosis by cytologic study	47
Diagnosis by bronchoscopic biopsy	24
Diagnosis by deformity, stenosis, etc.	15
Bronchoscopy positive, secretions negative	4
Bronchoscopy negative, secretion positive	12

While the differences between the percentage rates of 82.4 for the cytologic diagnoses and 68.4 for the bronchoscopic diagnoses is impressive, of more importance is the group of 12 cases, or 21 per cent, in which bronchoscopy was negative and cytologic examination was positive. It is evident that there is no need for a cytologic diagnosis when a positive bronchoscopic biopsy is obtained. It also is recognized that in a large number of cases of bronchial stenosis with deformity and fixity the bronchoscopic diagnosis of carcinoma is correct. A morphologic diagnosis, however, is desirable. In the above-noted 12 cases there were no positive bronchoscopic findings to suggest carcinoma.

REPORT OF CASES

CASE 1.—Male, age 60 years. He gave a history of cough and mucoid expectoration of five weeks' duration following a cold. Recently the sputum was streaked with blood. Roentgen study revealed

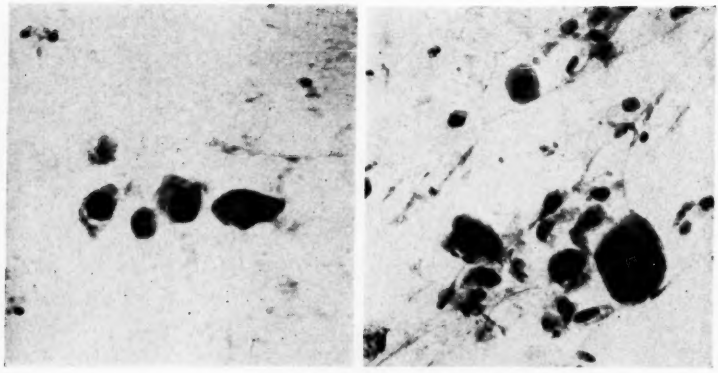

Fig. 3.—Smear of bronchoscopically removed secretions showing three cancer cells. The cytoplasm stains grey and the nuclei are relatively large and very deeply stained. Papanicolaou stain. x 340.

Fig. 4.—Smear of bronchoscopically removed secretions showing at least seven cancer cells. They vary greatly in shape and size, the cytoplasm stains a dirty grey and the nuclei are irregular and deeply stained. Papanicolaou stain. x 340.

changes in the left lung (Fig. 1). At bronchoscopy the findings were negative. A small quantity of blood-tinged secretion was secured from the orifice of the left upper lobe bronchus for cytologic study. A diagnosis of carcinoma was made (Fig. 3). Surgical exploration was urged. For unaccountable reasons nothing was done until over four months later when a report was received from another clinic advising that there was present a pleural effusion containing malignant cells with bronchoscopic evidence of extrinsic pressure on the bronchi on the left side but no tumor was visible.

Comment.—It is very probable that successful surgical treatment was possible when a positive diagnosis of carcinoma was made by cytologic examination of bronchoscopically secured secretion four months previously.

CASE 2.—Male, age 53 years. This patient was an excessive smoker of cigarettes and gave a history of cough of long duration. Recently there occurred hemoptysis, weight loss and night sweats. Roentgen study revealed a lesion in the right lung (Fig. 2). At bronchoscopy slight bleeding was encountered in the right lower

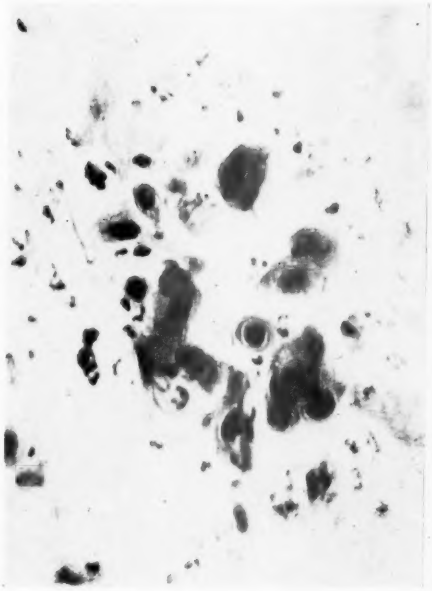

Fig. 5.—Smear of bronchoscopically removed secretions showing numerous cancer cells. They vary in shape and size, the cytoplasm stains a dirty grey to pink or orange, and the nuclei are intensely hyperchromatic. Papanicolaou stain. x 400.

lobe bronchus. This was traced to its apical subdivision. There was no evidence of neoplasm, ulceration, stenosis, fixity or deformity. Secretions were secured for cytologic study. These contained cancer cells (Fig. 4). Surgical exploration was advised and a right pneumonectomy was performed. A carcinoma was found in the lower lobe of the right lung not in close proximity to the lower lobe bronchus.

Comment.—The bronchoscopic findings were negative. The location of the growth in the lower lobe suggested that changes affecting the bronchus would not have occurred during its operable stage.

The occurrence of slight hemoptysis may interfere with the successful completion of a bronchoscopic study. It now is our practice

Fig. 6.—Histologic section of the tumor in the left bronchus showing a transition from normal epithelium to cancer tissue. The latter although typically squamous cell carcinoma has not yet penetrated into the submucosa. Hematoxylin and eosin stain. x 50.

to secure material for cytologic examination in all suspicious cases of neoplasm. The following case is illustrative:

CASE 3.—Male, age 60 years. He gave a history of cough and expectoration of ten years' duration. For the past three months there was wheezing respiration and pain in the chest. A roentgen study of the chest was negative. At bronchoscopy blood-tinged secretion was observed in the left bronchus. Removal of a specimen of secretion increased the bleeding and the bronchoscopy was discontinued. Cytologic study revealed cancer cells (Fig. 5). A second bronchoscopy was performed three days later. There was no bleeding and a superficially ulcerated lesion was observed in the posterolateral wall of the left lower lobe bronchus. A biopsy was done. The histologic diagnosis was carcinoma. Left pneumonectomy was successfully performed. The carcinoma appeared as a superficial ulcer

involving the posterior and lateral walls of the bronchus (Fig. 6). There was no metastasis.

Comment.—While the carcinoma was visible by bronchoscopy, a cytologic diagnosis was possible several days in advance of the histologic diagnosis. An ulcerated lesion "around the corner" could have been diagnosed with the same degree of certainty.

SUMMARY

A diagnostic aid is presented to supplement the procedures already employed in the diagnosis of bronchogenic carcinoma. This must not be confused with studies of sputum.

It is of greatest value as an adjunct in cases which exhibit suggestive bronchoscopic evidences of carcinoma and in those presenting roentgen evidences of pulmonary disease simulating carcinoma but which are completely inaccessible to bronchoscopic visualization. It is believed that, when more frequent roentgen examinations are made and all patients with questionable shadows are subjected to bronchoscopy so that secretions may be secured for cytologic study, the diagnosis of bronchogenic carcinoma will be made earlier, more patients will be found suitable for surgical treatment and the prognosis of bronchogenic carcinoma will become more hopeful.

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LXI

INJURIES TO THE TRACHEA AND THE ESOPHAGUS INCURRED IN COMBAT

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Most doctors in the service during the war have seen many and varied types of combat-incurred injuries involving the neck and the thorax. Freak explosions, blast and concussion have all taken their toll.

The following case was intriguing to us in that the upper trachea was the site of a severe cicatricial change following the patient's exposure to a terrific explosion.

REPORT OF A CASE

R. A. M. S1/C USNR, age 20. He was first admitted to the sick list at a naval hospital in California on November 26, 1945, with an undetermined diagnosis (asthma). On July 25, 1945, during the Philippine Islands liberation, he was injured in an explosion which occurred in the living quarters in the fantail of his destroyer. After he recovered consciousness he crawled to the ladder and onto the deck. The explosion, probably secondary to a below water torpedo attack, was very intense, and the irritating gas and steam fumes were so thick that he could see only about one foot ahead. He was very short of breath, his eyes were swollen shut and his mouth, throat and larynx were very sore. Two shipmates were killed outright and two others died shortly afterward. Eighteen others were injured, and the patient knows of one survivor who has had similar laryngeal trouble. He could not make any sounds for two or three weeks and was hoarse for three weeks longer. There was no sense of smell or taste for three weeks. Carbon tetrachloride fumes exposure is recorded in his health record. He does not remember if there were any sulphur fumes but states that there could have been. He was transferred to a middle western naval hospital January 1, 1946, the diagnosis having been determined as stenosis of the trachea. At this activity he was first seen by us. A cicatricial stenosis about the size of a slate pencil could be seen one inch below the larynx, with the laryngeal mirror. Lateral cervical roentgenogram showed

Fig. 1.—Tracheal stenosis. Arrows point to area of marked cicatricial change.

almost complete stricture of the trachea at the level of the second and third tracheal rings. Efforts at direct laryngoscopic dilatations were unsuccessful because of the firmness and extensiveness of the stenosis. His dyspnea progressed and a tracheotomy was performed. Two weeks later a tracheostomy was done and approximately one square inch of skin was transplanted and sutured to the posterior tracheal wall after the removal of the cicatricial tissue. A core mold, made of dental composition, was placed in this area to maintain the position of the transplant. Ten days later the core mold was removed and examination revealed that most of the skin graft had maintained its position. An acrylic obturator, made according to the technic outlined by Erich,[1] but hollowed to allow the passage of air into the pharynx in order that the patient could cough and phonate, was placed in the trachea and it was worn for three months. During this period this man has been able to resume most all of his athletic activities with the tracheal cannula corked. On removal

of the acrylic tube it was most striking to note the cleanliness of the inner and outer walls of the hollowed tube. Also the tracheal mucosa appeared healthy, showing no ill effects of the long residence of the foreign body dilator. The silver tracheal cannula also was removed and at the last report the patient was breathing normally.

The toxicological picture of poisonings from inhalation of various fumes has been studied. However, in the case that we have presented, the formation of irritating gas was the result of explosion in a closed room and not from any gas warfare.

Poisonings from exposure to nitrous fumes have been reported repeatedly. The incidence of fatalities in such accidents appears to be high, especially if high explosives are burned or exploded in the absence of sufficient air. The most serious and characteristic effects of nitrous dioxide fume poisoning refer to the respiratory apparatus. There are some irritant effects, such as choking sensations, pain in the throat and the chest, and cough. However, the irritant action is not in proportion to the injurious effects on the lung. Not infrequently this gas is inhaled freely without causing a prohibitive reflex such as laryngospasm. The most common sequela is pulmonary edema which develops after a latent period of several hours and which, in a great number of cases, ends fatally in one to three days. Bronchiolitis obliterans is described as a sequela, and Cramer[2] points out that inflammatory reactions are more severe in the smaller bronchi than in the upper sections of the bronchial tree. Nitric acid may be formulated by the following reaction:

$$3NO_2+H_2O=2HNO+NO$$

The thermal decomposition of nitrocellulose films may give rise to serious accidents, as illustrated by the Cleveland Clinic disaster in which fatalities were presumably partly cause by inhalation of carbon monoxide, hydrocyanic acid and nitrogen oxides.

In fairly high concentration sulphur dioxide is corrosive, forming sulphuric acid on combining with water. Its destructive action on moist surfaces of mucous membrane is thus explained.

Tracheobronchial mucosa being ciliated columnar epithelium is more vulnerable to the destructive effect of poison gases. All articles reviewed discuss these effects but none reports cicatricial changes in the location and to the extent that our case presents.

Gordon,[3] reports a case of partial left bronchial stenosis following inhalation of sulphur dioxide fumes. Von Oettingen[4] states

that the main effect of sulphur dioxide is an irritation of the upper respiratory tract, but it may cause lesions of the bronchi and lungs.

Haggard[5] states that all respiratory irritants have a similar toxic action. The difference in symptomatology varies with the location of their action and is dependent on the relative solubility of the irritating gases.

Supposedly sulphur dioxide gas is an irritant that is irrespirable but actually it can be inhaled by acclimatizing or acquiring the ability to withstand the customary basic exposure. A significant higher incidence of nasopharyngitis, both chronic and slight, of alteration in sense of smell and taste, of increased sensitivity to other irritants was found in a group of men exposed to sulphur dioxide.

Sulfonamide therapy is recommended for acute sulphur dioxide poisoning by Goldburgh.[6]

Romanoff[7] reports three cases of bronchial asthma following repeated exposure to sulphur dioxide from leaking refrigerators. Bronchoscopic examination was performed in one case revealing a large amount of cheesy secretion in both bronchi. He recommended that individuals with an allergic background should avoid exposure to sulphur dioxide.

The outstanding pathological feature in the lungs in acute phosgene poisoning is pulmonary edema according to Gilchrist.[8]

After gassing with phosgene, there is some irritation of the trachea and bronchi. Coughing is not a prominent symptom and disruptive emphysema is practically never seen. After moderate gassing a man may feel able to carry on his work for an hour or two and then suddenly become worse. The increased capillary permeability and the intense acid reaction result in development of pulmonary edema. The epithelium of the trachea and the bronchi is not damaged. The more distal portions of the lung suffer the most. Chronic bronchitis is a frequent residual of phosgene poisoning.

The chemical reaction whereby carbon tetrachloride can be converted into phosgene is as follows:

$$2CCl_4+O_2=COCl_2+2Cl_2$$

During World 1, phosgene was made by the combining of CO and Cl_2 in the presence of charcoal.

The arsenicals are similar to chlorine and phosgene in their physiological action on the respiratory tract, resulting in congestion

of the nose, throat and bronchi and edema of the lungs. Mustard gas will destroy the epithelial lining extending to the small divisions of the bronchial tree.

The diagnosis and symptoms of tracheal injury are closely associated. The respiratory rush of air, with bloody froth through a gaping wound, dyspnea, cyanosis and cough are all dependent on the location and extensiveness of the wound. Subcutaneous emphysema and hemorrhagic infiltration frequently occur and may progressively increase. Bronchoscopy and clarification of diagnosis is indicated, often affording immediate relief of the dyspnea. Nach and Rothman[9] have recommended multiple incisions through the skin and the subcutaneous tissues of the neck and the use of a large rubber tube placed in the trachea to relieve the associated mediastinal emphysema. The danger in the use of morphine under such circumstances is emphasized.

The author has had no actual combat experience, but has cared for many of these combat victims on their stop on the mainland. This paper would not be complete without recording in detail the activities of advance units during an active invasion period. I am particularly indebted to two of my friends, fortunately trained endoscopists who were attached to the U. S. Marine Corps and engaged in four of the major Pacific island invasions, for a description of actual combat conditions. They make statements of conditions of which I have no personal knowledge.

The following are the observations of Commander Claude R. Bruner[10] (M.C.) U.S.N.R., of casualties of one combat division.

"During the initial combat stage, from D-day until D-plus-2 day, all casualties were evacuated to ships after receiving first aid and shock treatment at beach evacuation stations. On the afternoon of D-plus-2 day one medical company established a hospital in a damaged Japanese hospital and from that time until D-plus-6 day approximately 50% of all casualties were evacuated through this installation and did receive some definitive care. On D-plus-6 day, the division hospital was set up and from that time on all casualties were given some definitive care before evacuation. Naturally, on the first two days very little could be done in a definitive way for casualties involving the respiratory system. From D-plus-2 day until D-plus-6 day, it was possible to give some valuable definitive care to this type of casualty. From D-plus-6 day until hostilities ceased there was an opportunity, had the equipment and personnel been available, to have given quite adequate definitive treatment.

As neck and chest wounds are particularly prevalent in the initial stages of a landing operation and movement inshore, it is fair to estimate that this type of casualty is even greater than in later periods when the troops have more chance to protect themselves in their advance. Based on the figures to be given, it is a reasonable estimate, that as many as 50 wounds of the respiratory structures were without adequate initial care on each of the first two days following the landing and 25 of such type cases received inadequate care on the following four days.

"Exact figures in the first hospital installation, from D-plus-2 day until D-plus-6 day, show two neck wounds requiring tracheotomy and 15 sucking chest wounds treated. After the division hospital was established on D-plus-6 day, the figures show that of 2,098 combat wounds, there were 72 intrathoracic wounds, 13 chest and abdominal wounds, 17 wounds of the chest and extremities and 27 wounds of the neck involving either the pharynx, larynx or trachea. Of this total number, there were 27 deaths after these patients were received in the hospitals. In at least 50% of all these cases one form of endoscopic treatment was indicated and necessary to properly evaluate the nature and type of the wounds, and in at least 25% of the cases it was the only means of saving the patients. Wounds involving the upper respiratory structure frequently caused an obstruction to the air passage, either by swelling, hemorrhage or displacement of solid structures plus the inevitable accumulation of secretions. Thus, it is readily seen that restoration and maintenance of the airway is often the deciding factor in any given case.

"The only instruments available on the operation for treating this type of casualty was an antiquated laryngoscope, a seven-millimeter bronchoscope without a light and with improvised aspiration tubes. These instruments were available only because I begged for them at the supply depot in Pearl Harbor. Of course, we did have tracheotomy tubes. In addition to this I utilized two queer Japanese bronchoscopes, which I found in the abandoned Japanese hospital.

"On the hospital ships endoscopic equipment is available. However, the hospital ships do not arrive for a few days and some ships did not have an endoscopist aboard. Therefore it is reasonable to state that in modern combat medicine, there should be at least a minimum of essential instruments and trained personnel with the combat ground forces to provide at least the same degree of initial care in this type of wounds as is provided for wounds of the abdomen and extremities."

Statement of Thomas L. Smith, Lt. (M.C.) U.S.N.R.[11]

"During my tour of overseas duty I was attached as otolaryngologist to a Marine Corps Medical Battalion. The organization was equipped as a 1,500 bed field hospital, assigned to carry out the first definitive treatment of casualties, and later evacuate the patients to the rear areas for further protracted treatment and convalescence.

"During a major campaign, extending from April 1, 1945, to June 21, 1945, we actively cared for slightly over 7,000 casualties. Of these, I recall eight or nine cases of severe traumatic injury, by gunshot or shapnel, to the throat and larynx. Although not part of our battalion's accredited table of supplies, I was fortunate to have acquired, prior to the beginning of the campaign, a laryngoscope and a bronchoscope which I operated from a two-cell diagnostic kit battery handle. In each of the throat injury cases the use of the laryngoscope and the bronchoscope was of paramount importance, or at most, it was a life saving procedure. Most of the patients were moribund on arrival. Blood, which was profuse, was being aspirated into the bronchial tree, together with other secretions."

In discussing the various types of combat incurred injuries to the neck and thorax with several of my colleagues, the need for a trained peroral endoscopist and sufficient equipment near to the front line activities has become apparent. Many lives could be saved by a prompt and judicious bronchoscopy. The amount of equipment would involve a laryngoscope, one or two bronchoscopes, one or two forceps, a small battery, and aspirating tubes.

For this paper there is not sufficient time to completely discuss all phases of trauma and care of the larynx, trachea and esophagus incidental to war injuries. Jackson and Jackson[12] have a very excellent chapter in their textbook which should be studied. Lederer and Howard[13] also discuss various types of wartime laryngeal injuries.

DeVilliers[14] recommends the use of a long metal tracheal cannula for stenosis of the trachea. In external frontal sinus operations, the use of the acrylic tubes to maintain a patent nasofrontal opening has proved satisfactory. The apparent ease with which the tube "maintains its own cleanliness" is a recommendation over all other types of metal or rubber cannulas or obturators and accordingly the author recommends that a set of longer acrylic tracheal cannulas should be available for use in the field for cases of tracheal injury, especially where compression from emphysema precludes the value of the shorter standard sized silver tracheal or tracheotomic cannulas. This

cannula should have the advantage of not requiring a specialist's attention for the several days that it might have to be worn while the patient is awaiting definitive care. Experimental animal studies should be performed to verify this point.

The author has used a rubber tube with transfixing silver wire as recommended by Von Schmiegelow[15] for laryngeal stenosis. There appeared to be a marked tissue reaction to the rubber. Vitallium and tantalum dilators could be used. However, the accessibility of dental laboratories and the value of being able actually to fit the acrylic obturator plus the tolerance which the tissues have for this foreign body, make one strongly inclined to the Erich method. The suturing of the skin transplant, the use of a temporary hollow core mold and a tubular acrylic mold were the only variations in technic.

REPORT OF A CASE

R. A. P., Y1/C U.S.N.R., age 23. When the USS ..... was sunk in July 1945 this patient was in the water for five days before being picked up by a destroyer. A kapok life jacket was his means of survival. During these five days he swallowed a great deal of sea water and oil. He was admitted to the sick list on August 4, 1945, at a naval base hospital in the Western Sea Frontier, with the diagnosis of contusion of the back. He also suffered from overexposure, many skin ulcerations, and a large deep ulcer over the left trochanter, which was cured at a later date by split thickness skin graft. There was also paralysis of the right radial nerve, which was secondary to his holding his knees with his hands while he was attempting to get some sleep when in the water. He recovered from this condition within two months. On August 6 he was placed aboard a hospital ship and a diagnosis of "esophagitis and gastritis, chemical, brine and fuel oil," was made. Chest x-ray studies showed persistent narrowing of the lower half of the esophagus without apparent mucosal pattern or actual filling. A diagnosis of "esophageal spasm, etiology not apparent" was made.

During his four weeks' stay at this hospital he was having considerable difficulty in swallowing liquids and failed to respond to belladonna and phenobarbital medication. An esophagoscopy at the earliest possible date was recommended. He was fed by Levine tube for 72 hours. Just before leaving, the tube was removed and the patient was able to swallow liquids better. He was evacuated to the mainland by ship, which passage required three weeks. During that time he continued to have difficulty in swallowing.

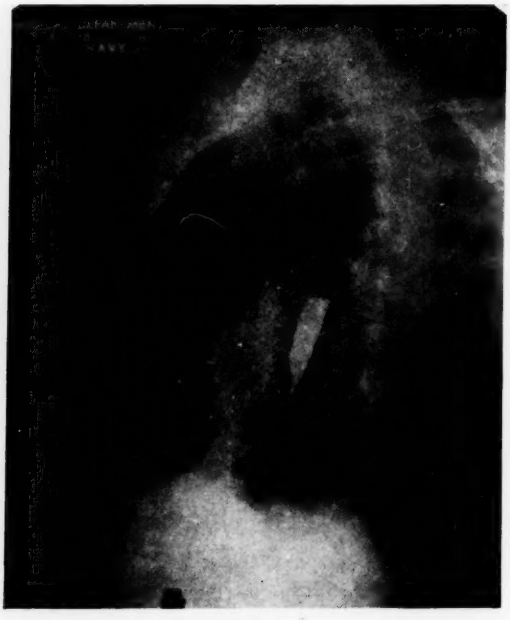

Fig. 2.—Atresia of Esophagus. Note area of complete obstruction in lower third of the esophagus.

His admittance diagnosis of spasm of the esophagus was changed to esophagitis on November 5, 1945, following an esophagoscopy, during which acute inflammatory stricture with membrane formation which bled easily was observed. A specimen of the esophagus showed a pyogenic membrane. Three esophagoscopies were performed in the following three weeks and aspergillus niger was cultured from the hypertrophied membrane. Five weeks of penicillin therapy by intramuscular administration apparently helped his discomfort and corrected the ulcerated lesion. A concentric cicatricial narrowing of the esophagus was observed on November 15. Three dilatations were performed under direct vision in the following month before he was transferred to a middle western hospital on December 24, 1945.

Previous to his transfer here a gastrostomy had been recommended by one of his doctors. However, no one had recommended

that he swallow a string. He had gained approximately seven pounds from his low of 120 pounds, but was still below his normal weight of 170 pounds. He was unable to swallow any fluids, or even saliva, at this activity, and a gastrostomy was performed on January 9, 1946. He was first seen by our department on January 18, seven days after the operation.

He gave a history of ingesting two dry biscuits from a K ration on his fourth day in the water. These rations were dropped by a PBY rescue plane. The biscuits seemed to stick in his throat and he "couldn't get them down." When he was rescued he found that swallowing food, or even liquids, caused a burning sensation and soreness and for two days he vomited small amounts of oil. Attempts to swallow solid foods would result in retching and vomiting, and his substernal pain associated with swallowing persisted for at least two months.

One week after the gastrostomy a retrograde esophagoscopy was performed and a 12F Jackson soft nose esophageal bougie was passed through the atresia and a string was recovered in the pharynx. This made a total of approximately 20 days that he had been completely atresic. A series of retrograde dilatations with Tucker bougies were performed and the patient gained 36 pounds in the following 40 days. The gastrostomy tube and continuous string were removed on March 14, after a 40 French Tucker retrograde bougie had been satisfactorily passed on three previous occasions. His weight at this time was 162 pounds. His general condition was excellent.

Most likely the cause was a foreign body impaction, complicated by dehydration and swallowing of sea water and oil. From the patient's own story he had at least four days of complete obstruction which apparently was sufficient to cause a very extensive esophageal ulceration.

SUMMARY

1. The acrylic obturator method, as outlined by Erich, is recommended for surgical correction of tracheal stenosis. A variation or modification of technic was used in the reported case.

2. Esophageal stenosis and even atresia can result from impaction of food matter. The swallowing of a guide string before there is complete closure, even in combat cases undergoing transportation, should always be kept in mind.

3. It is hoped that from the lessons learned in this war that provisions will be made for more adequate personnel and equipment for the treatment of combat-incurred respiratory wounds, at least to the same degree to which care is provided in other types of wounds.

4. A suggestion is made for the development and the emergency usage of longer tracheal cannulas to be made of acrylic resins. Such cannulas would be of value in civilian accidents.

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LXII

PERFORATION OF THE ESOPHAGUS NOT CAUSED BY INSTRUMENTATION

REVIEW OF EIGHT CASES

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Although perforation of the esophagus is of relatively rare occurrence, it is always a potentially serious condition requiring the best judgment and skill in its management. Hence it seems worth while to report a series of cases of this type which presented some unusual features as to etiology and treatment. Four of the eight cases were observed personally, and the others are included in this group through the kindness of my colleagues, F. W. Dixon, J. W. McCall and Horace E. Mitchell. Although cancer is generally cited as the most common cause of esophageal perforations,[1, 2] it is interesting that none of these cases was of this origin. Foreign bodies are considered to be the second most frequent cause, and six of these cases were due to ingested foreign bodies. The remaining two resulted from a caustic burn (ingested lye) and from syphilis; in both these instances there was formation of a broncho-esophageal fistula.

The first two cases are interesting from the standpoint of treatment in that both patients recovered with conservative treatment after a relatively short period of approximately two weeks in the hospital. Furthermore, both these patients were observed before sulfonamides or penicillin were in general use. Although conservative treatment was successful in these instances, that does not mean that they are presented as a recommendation that all patients should be so treated. In these cases, the symptoms indicated that the infection was localized and there was no evidence of rapid progression. They undoubtedly belonged to the first of the two major groups designated by Holinger,[1] i.e., they were examples of localized infections of periesophageal tissues, characterized by fever, pain and difficulty in swallowing. The systemic symptoms were moderate, even though the local manifestations were relatively severe. Holinger noted that spontaneous healing occurs relatively frequently in this group. Many authors stress the necessity for surgical treatment in practically all

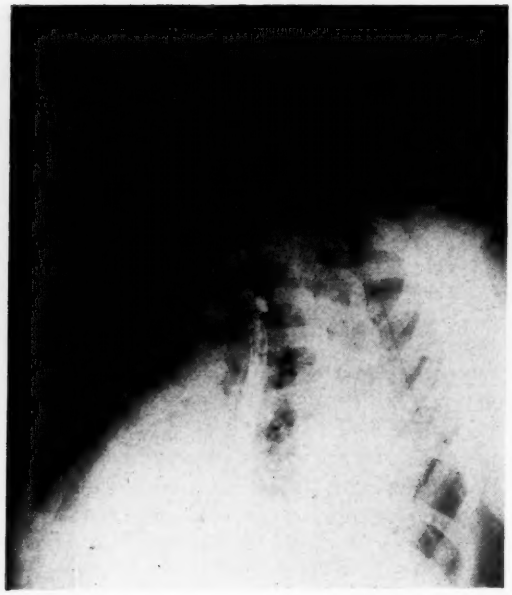

Fig. 1.—Roentgenogram, oblique view, in Case 1 on admission, showing barium passing freely through the esophagus, with a small quantity apparently entering a passage posterior to the esophagus, from a point just below the inferior border of the larynx to 4 cm. below the clavicles.

cases of esophageal perforation. Early external drainage is advocated to prevent mediastinitis by Grace and Irwin[3] and by Pearse.[4] Holinger advocates immediate external drainage in any case in which there is an inflammatory process advancing through the retroesophageal tissues or the mediastinum.

REPORT OF CASES

CASE 1.—A woman, aged 35 years, gave a history of having swallowed a part of the breast bone of a duck one week before admission to St. Vincent's Charity Hospital. A roentgenographic examination made at the time of the accident by her family physician failed to reveal the presence of a foreign body. Later she had suffered severe pain, localized in the lower part of the sternum, with radiation toward the back. This pain was aggravated by swallowing.

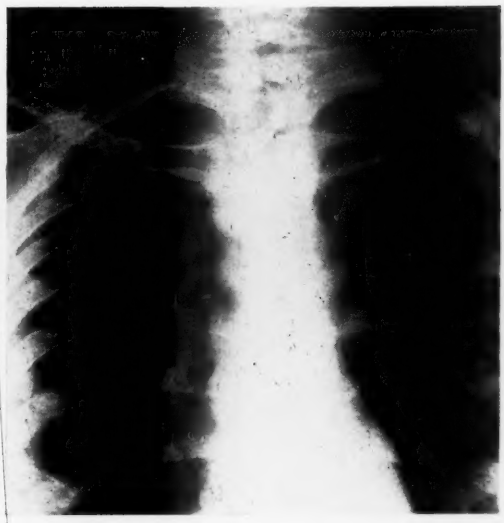

Fig. 2.—Roentgenogram, anterior-posterior view, in Case 1 nine days after admission, showing slight widening of the mediastinal shadow.

On admission, the patient's temperature was 38.7° C. Physical examination yielded essentially normal findings. Fluoroscopic studies and roentgenograms of the esophagus showed that barium passed freely through the esophagus, but a small quantity apparently entered a passage which lay posterior to the esophagus. This passage was visualized from a point just below the inferior border of the larynx to 4 cm. below the clavicles (Fig. 1). Esophagoscopic examination was performed, and no foreign body could be found; nor could the small laceration of the esophagus be visualized. Just beyond the cricopharyngeal narrowing there was considerable swelling of the esophageal wall, owing to submucous hemorrhage.

The patient's diet was restricted to boiled liquids. Fluoroscopic examination was done 48 hours after the esophagoscopy and showed retention of barium in the sinus tract at a point 4 cm. below the suprasternal notch. The patient's temperature ranged from 38.7° C. on the day of admission to 39.6° C. on the fifth hospital day. After this, the fever gradually subsided, and the temperature returned to normal.

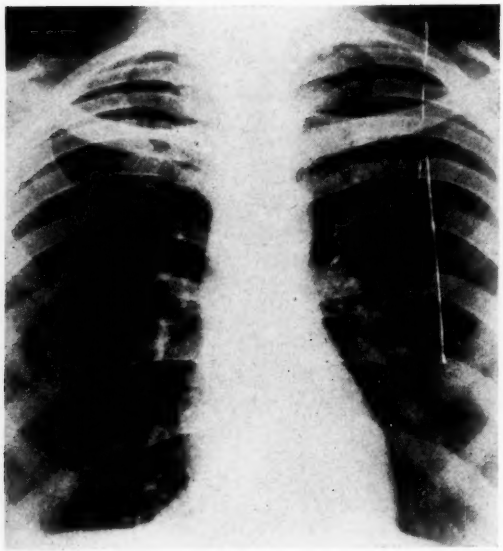

Fig. 3.—Roentgenogram, anterior-posterior view, in Case 1 about three weeks after that in Fig. 2, showing that the slight bulging of the mediastinal shadow at the sternal notch had disappeared.

Nine days after admission, roentgenologic examination of the chest showed that the lungs were clear and that both domes of the diaphragm moved normally. The sinus tract was still visible and extended to the same level as at the previous examination. There was also a large irregular cavity in the prevertebral space, situated at or just above the level of the sternal notch. A plain roentgenographic film of the mediastinum showed a slight bulging of the mediastinal shadow (Fig. 2). There was also a linear shadow 5 mm. long in the posterior mediastinum at the level of the sixth dorsal vertebra. This shadow was probably caused by some opaque substance retained from the previous examination rather than by an actual foreign body, since it was not demonstrable on any other roentgenograms.

Aside from bitter complaint of hunger, the patient made satisfactory progress, even though a fetid breath, slightly blood-tinged sputum and some substernal pain persisted for one week after admission. Three weeks later, another plain roentgenogram showed no

evidence of an opaque foreign body in the posterior mediastinum, and the previously described slight bulging of the mediastinal shadow at the level of the sternal notch was no longer present (Fig. 3). The sinus tract, which in earlier roentgenograms appeared to be about 12 cm. long, was not more than 3 cm. long. The cavity in the prevertebral space was no longer present.

After the patient was discharged from the hospital, additional fluoroscopic examinations were made periodically, and finally there was complete obliteration of the sinus tract.

Comment: When this patient was admitted, she was complaining of pain radiating to the back. This is a common symptom in cases of esophageal perforation and has been cited by Holinger,[1] Pearse[4] and Robson.[5] The fetid breath, blood-tinged sputum and substernal pain, present during her hospital stay, are also common symptoms.[1, 2, 6] It would appear that this patient may have had some constitutional protective mechanism which prevented or controlled the infection. Hayes[6] has commented that probably an inflammatory process produces a protective barrier about the perforation or lesion. Holinger has noted that there is tremendous individual variation in response to such an injury and that is why careful evaluation and individualization of cases is required. As already mentioned, this patient did not have the benefit of modern chemotherapeutic agents, and she also had a perforation of the posterior esophageal wall, which, in Pearse's opinion, is more likely to cause mediastinitis than is a perforation of the anterior or lateral walls.

CASE 2.—A woman, aged 50 years, had felt a bone lodge in her throat, while eating chicken, three days before admission to City Hospital. She attempted to force it down by eating bread but this was unsuccessful. Since then, she had been able to swallow only water. Her symptoms consisted of severe pain at the level of the cricoid cartilage, which was more intense on swallowing, and hoarseness. For 36 hours before she entered the hospital, she also had had pain in the right ear.

When she was examined, her temperature was 39° C. and the leukocyte count was 11,450. Examination of the larynx showed pronounced edema of the right arytenoid cartilage. Roentgenologic examination revealed a perforation in the posterior esophageal wall at the level of the cricoid cartilage (Fig. 4). One-half a chicken wishbone was present in this region, with one end extending through the posterior esophageal wall; a pocket of air was present above it (Fig. 5). In addition to the pronounced edema of the right ary-

Fig. 4.—Roentgenogram, lateral view, in Case 2 on admission, showing an esophageal perforation in the posterior esophageal wall at the level of the cricoid cartilage, with the barium extending through it to the vertebral process.

tenoid cartilage, esophagoscopy showed that swelling of the esophageal wall was so great that it had almost completely obscured the foreign body. The edematous esophageal wall was pushed aside until the head of the wishbone, which pointed upwards and was situated 3 cm. below the arytenoid cartilage, could be seen and grasped. The foreign body removed consisted of the head and about two-thirds of the crura of the chicken wishbone.

The day after esophagoscopy was performed, the patient's temperature was 38.5° C. and gradually decreased to normal within a week. She received boiled liquids for one week, after which semisolid food was taken. She was discharged on the tenth hospital day.

Comment: This patient, like the first, was treated conservatively, and without use of sulfonamides or penicillin and she also recovered promptly and satisfactorily. In this instance also, the perforation was on the posterior wall of the esophagus and there was no extensive spread of infection into the mediastinum. The foreign body was situated high in the esophagus, accounting for the

Fig. 5.—Roentgenogram, lateral view, in Case 2 on admission, showing the foreign body, consisting of one of the crura of a chicken wishbone penetrating through the posterior esophageal wall, with a pocket of air above it.

edema of the larynx, the hoarseness and the radiating pain to the right ear. In neither of these patients was a feeding tube inserted, since is was feared that, owing to the marked edema of the esophageal tissues, the tube might be pushed through the perforation into the mediastinum.

The next two patients were observed by Dr. Fred W. Dixon, and I am indebted to him for the privilege of reporting them in this series.

CASE 3.—A white woman, aged 24 years, was admitted to St. Alexis Hospital three days after she believed she had swallowed a piece of glass, broken from a mustard jar. Since that time her throat had been swollen and painful and she had been unable to swallow.

When she was examined, her temperature was 103° F. Roentgenographic examination showed a pathologic process, containing

some air or gas, situated anterior to the vertebra behind the lower end of the pharynx and the upper end of the trachea. Esophagoscopy revealed no foreign body, but a laceration was visualized on the right side of the posterior wall of the esophagus. This bled slightly but no pus was seen. The leukocyte count was 14,800 and the sedimentation rate was 45.

Sulfanilamide in a dosage of 30 grains every four hours was administered. Fever persisted and two days after admission the leukocyte count was 20,400. The retro-esophageal space was opened on the right side. No pus was encountered, but a very foul fecal odor was present. Sulfanilamide powder was dusted into the space, which was drained with a Penrose tube, and the cavity was irrigated with Dakin's solution. Bacteriologic study of a culture of material obtained from the wound at operation showed anaerobic streptococcus. A Levine tube was used for feeding for four days. Roentgenologic examination six days after esophagoscopy showed considerable reduction in the swelling in the retro-esophageal space.

Comment: In this instance, the increase in leukocytes from 14,800 to 20,400 during the first two days after admission, despite administration of adequate doses of sulfanilamide, furnished a definite indication for mediastinotomy. After this procedure, the symptoms subsided and the patient made a completely satisfactory recovery.

CASE 4.—A male infant, aged 10 months, while playing on the floor where several loose thumb tacks lay, had placed one in his mouth and immediately began to gag. A little later, he vomited some bloody mucus and then was brought to the hospital. Roentgenologic study revealed a tack in the upper end of the esophagus at the level of the fifth cervical vertebra. The point of the tack extended posteriorly; the head was situated anteriorly. The esophagoscope was passed without anesthesia, the head of the thumb tack was easily visualized and removed without difficulty, although rather firm traction was required to dislodge it. No bleeding accompanied this procedure.

The day after esophagoscopy was performed, the patient's temperature rose from 101° F. to 105° F. and on the following day to 106° F. The leukocyte count was 20,000. Roentgenographic examination made at this time showed that the pharynx, larynx and trachea were displaced forward and that gas was present posterior to the upper end of the trachea. The leukocyte count had risen to 29,700 and drainage of the abscess cavity in the mediastinum was

carried out at the level of the fifth and sixth cervical vertebrae on the right. Bacteriologic examination of cultures from the wound showed Staphylococcus albus and Streptococcus anhemolyticus.

Sulfapyridine, grains 5 every four hours, was administered. Considerable drainage persisted for three days, during which time the leukocyte count was reduced to 11,000. By the fourth day after operation, discharge from the neck wound had ceased. The patient was discharged from the hospital on the tenth postoperative day with a normal temperature and a leukocyte count of 10,700.

Comment: This case also points up the value of the leukocyte count in determination of proper treatment. After removal of the foreign body, the white blood cell count soared to 29,700, thus indicating the necessity for drainage of the mediastinal cavity. Within three days after this procedure, the leukocyte count dropped to 11,000. With such a small perforation from a tack point, one might expect that the inflammatory process might produce a protective barrier to extensive spread of infection, thus permitting conservative treatment. Clinical and laboratory findings indicated that this did not occur; hence it was necessary to drain the mediastinal abscess.

The following case is included through the courtesy of Dr. Horace E. Mitchell:

CASE 5.—Four days before her admission to Lakewood Hospital, a woman, aged 54 years, had experienced a sharp pain in her throat while eating chicken pie. She had suspected that she had swallowed a bone and consulted several physicians who concluded that she had an infection in the throat and prescribed the usual gargles, sedatives and also sulfathiazole. When Dr. Mitchell examined her, she had pain in the throat, difficulty in swallowing, and the right pyriform sinus was filled with mucus.

Roentgenograms revealed only a minimal increase in width of the mediastinal shadow. On esophagoscopic examination, a laceration was found on the right side of the esophagus. The foreign body, part of the breastbone of a chicken, triangular in shape and very sharp, measuring approximately an inch on each equilateral side, was removed without difficulty.

The usual regimen for perforation of the esophagus was instituted: withholding of food, and intravenous administration of glucose and sodium chloride solutions and of sulfathiazole. On the sixth day after removal of the foreign body, the patient complained of pain in the chest and the back. Roentgenograms showed fluid

in the right pleural cavity. A chest surgeon was called in consultation and performed a thoracocentesis in the right eighth interspace, near the posterior axillary line. A thin, yellow-brown, turbid fluid (350 cc.), containing occasional gram-positive cocci, was obtained. Repeated thoracocenteses were done, with institution of intercostal drainage which yielded approximately a liter of thin pus.

Feeding through a Levine tube was carried out for one month. During this period, several transfusions were administered because of reduction in number of erythrocytes and in quantity of hemoglobin. A little over a month after admission, rib resection was done because the empyema was not draining well. Two months after this procedure, the patient was afebrile and the chest roentgenogram showed progressive expansion of the lung. After a hospital stay of 111 days, the patient was discharged, even though empyema persisted. She was followed for more than a year and made a complete recovery with no untoward sequelae.

Comment: This patient's course indicates the potential seriousness of every case of esophageal perforation. Although she eventually recovered completely, with no residual complaints, this was not accomplished without multiple surgical procedures, a long period of hospitalization, and severe morbidity.

Dr. Julius McCall observed the following case, and has kindly allowed me to report it in this group.

CASE 6.—A man, aged 24 years, was admitted to St. Luke's Hospital shortly after he had swallowed an upper denture. He was having severe pain in the neck and chest and was expectorating blood. Under local anesthesia, a short Mosher esophagoscope was introduced into the postcricoid region. The plate of the denture was seen as a hard brown object, and some bleeding was evident. It was decided that the denture could not be removed without severe damage to the esophagus. Later in the day, a general anesthetic was administered and the esophagoscope was again introduced. The sharp edge of the denture could be seen in the upper third of the esophagus; the teeth and double-pronged gold clasp on the denture were not visualized because they were hidden below this edge. The gold clasp was known to be lying on the left and was evidently protruding through the left esophageal wall. With rotation forceps the right side of the presenting edge of the denture was grasped and rotated upward to the left, thus pulling the teeth up into the right side of the esophagus. The entire denture was withdrawn, retaining the teeth in the right lateral position, with the gold-pronged points

dragging. Examination of the esophagus after removal of the denture revealed a ragged laceration in its left lateral wall where the clasp had perforated it.

On admission, the patient's temperature was 37.2° C.; the following day it had risen to 39° C., and the leukocyte count was 12,600. The day after the two esophagoscopic procedures both the anterior and the posterior mediastinum were drained. No free pus was elicited through the posterior approach, but a considerable quantity of pus was liberated when the anterior mediastinum was opened. Culture of this material showed Streptococcus alpha. The day after mediastinotomy, the temperature soared to 40.5° C. and the leukocyte count was 18,400. The patient died on the fourth day in the hospital.

Comment: Dr. McCall is of the opinion that if he were to be confronted with a similar case, he would remove the foreign body externally and drain the mediastinum at the same time. This case certainly falls into the second major group, according to Holinger's[1] classification, i.e., fulminating, extensive, overwhelming sepsis following esophageal perforation. Holinger states that such inflammatory processes are usually rapidly progressive and may involve the entire retro-esophageal space from the retropharyngeal area to the diaphragm in 24 to 36 hours. An interesting feature in this particular case was that, although the temperature rose to 40.5° C., the leukocyte count was only 18,400.

The following two cases are examples of acquired bronchoesophageal fistulas.

CASE 7.—A man, aged 35 years, was admitted to St. Vincent Charity Hospital after having swallowed lye in a suicidal attempt. He was treated by his family physician and one morning, three days after swallowing the lye, he coughed and spat up a large piece of tissue, measuring 10 cm. in length and consisting of a complete cast of the esophagus including the mucosa, the submucosa and in places the inner circular as well as the outer longitudinal muscle fibers.

Gastrostomy was performed, but the patient still insisted on taking some fluids by mouth, after which he would cough and bring up what he had swallowed. During this period the temperature would rise to 39° or 40° C. When barium was administered by mouth, fluoroscopic examination showed that it entered the left lung and delineated a definite picture of the left bronchial tree. The patient swallowed a string, and retrogade dilatation was instituted.

Inasmuch as he continued to cough and complained of pain in the chest accompanied by hemoptysis, bronchoscopic examination was requested and performed. A short distance below the carina in the left main stem bronchus, a white object was seen which proved to be the string coming from the esophagus through the defect in the left main stem bronchus.

The patient eventually left the hospital, but after he returned to his home achieved suicide by carbon monoxide poisoning.

Comment: Holinger[1] has stated that a caustic may produce an esophageal perforation by simple cauterization and erosion associated with widespread necrosis. Histologic examination of a section of this esophageal cast exhibited necrotic, highly edematous tissue, richly infiltrated by polymorphonuclear cells. The pathologic diagnosis was esophageal cast with necrosis of tissue and acute purulent inflammation.

CASE 8.—A woman, aged 47 years, was admitted to St. Vincent's Charity Hospital with the complaint of a lump in the middle of the neck, nervousness, palpitation, a weight loss of 20 pounds during the past year, and a paroxysmal cough productive of bright bloody sputum. She experienced some difficulty in swallowing foods and liquids.

Positive physical findings included symmetrical and diffuse enlargement of the thyroid gland with a slight bruit, pronounced pulsation in the suprasternal notch and over the carotids, marked precordial activity, irregular pulse. The abdominal reflexes were absent and others were sluggish. The liver edge was palpable. Her temperature was 40.2° C. and the Wassermann and Kline tests gave a 4 plus reaction.

Three days after admission, there were definite signs of consolidation in the left base posteriorly, with dullness and tubular breathing. The patient was expectorating a profuse amount of greenish sputum, which showed no tubercle bacilli. The temperature curve showed definite spikes. Roentgenographic examination of the chest showed an enlarged heart and also very pronounced basal trunk markings on the right with multiple small confluent zones of consolidation in the periphery of the left base. The roentgenologist concluded that these lesions were bronchopneumonia secondary to bronchiectasis. Additional roentgenographic studies two weeks after admission showed an increase in the bronchopneumonic process in the left base. Examination of the esophagus showed that some of the opaque sub-

stance was spilled into the larynx, from whence it entered the trachea and the bronchi. This could have been sufficient to explain the extension of the bronchopneumonic process. To preclude any possibility that the opaque substance would get into the trachea through the larynx, a catheter was inserted into the esophagus as far as the arch of the aorta. The opaque substance then moved not only down the esophagus into the stomach, but also directly into the tracheobronchial tree through a free communication with it a short distance below the level of the carina.

In view of these roentgenoscopic findings, bronchoscopy and esophagoscopy were performed. At 23 cm. from the gum line a clear hole was seen extending through the anterior wall of the esophagus. There was no piling up of tissue around this, no reaction and no sign of any tumor mass. Bronchoscopic examination revealed considerable whitish scarring about the lower part of the trachea and the openings into the right and left main stem bronchi. The carina was greatly thickened and rounded. Because of the scarring and narrowing of the tissues, it was necessary to replace the 9-mm. bronchoscope with a 5-mm. instrument. Even with this small scope, the left main stem bronchus could just be entered. It was impossible to visualize the fistula because of the marked contraction and narrowing of this bronchus.

Three days later, a gastrostomy was performed. The pathologic involvement of the lung increased, and the patient died three days after operation.

The principal findings at autopsy were broncho-esophageal fistula of the left main bronchus, bronchopneumonia of both lower lobes, bronchiectasis of the left lower lobe and syphilitic mesoaortitis.

Comment: The esophagus is not one of the common sites for attack by syphilis.

SUMMARY

In a series of eight cases of perforation of the esophagus, the cause was a foreign body in six instances. In the remaining two cases, there were acquired broncho-esophageal fistulas; one was due to cauterization by lye ingested with suicidal intent, and the other to syphilis. Of the six patients with esophageal perforations caused by foreign bodies, two were treated conservatively and experienced spontaneous recoveries; the other four were subjected to mediastin-

otomy, with recovery in three and death from a fulminating paraesophageal infection after removal of the foreign body (a denture) in the other. Both patients with broncho-esophageal fistulas died, the first after discharge from the hospital by suicide and the other from the disease. In all cases, roentgenographic examinations and endoscopy were important in establishing the diagnosis. Roentgenographic findings conformed to those previously described for this condition. Characteristic changes, as listed by McGibbon and Mather,[2] include: (1) a bubble of air or gas surrounding the point of a perforating foreign body in the cervical region; (2) forward or lateral displacement of the esophagus; (3) increase in depth of space between the bodies of the cervical vertebrae and the trachea; (4) widening of the mediastinal shadow in the anteroposterior view; (5) passage of barium from the esophagus into the tracheobronchial system; (6) opaque medium outside the esophagus. All these findings were demonstrated in this series. It is interesting that in two of the cases of esophageal perforation caused by a foreign body, the foreign body was not visualized roentgenographically nor esophagoscopically and was not removed (Cases 1 and 3).

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LXIII

ENDOLARYNGEAL SURGERY COMBINED WITH RADIATION IN LATE LARYNGEAL CANCER

MILLARD F. ARBUCKLE, M.D.

SAINT LOUIS, MO.

In this paper I wish to present an up-to-date report of my efforts at treating cancer of the larynx by a modified means of approach which I have devised. I am sure you will agree with me that laryngofissure,[1] as developed by Thomson, is the treatment of choice for cancer which is still localized to the true vocal cord and that total removal (laryngectomy) is generally accepted as the treatment of choice in cancer of the larynx with somewhat greater distribution. You will also agree, that when we are faced with the necessity of treating cancer of the larynx of widespread distribution we are at once confronted with a dilemma that presents the very greatest difficulties in so far as successful treatment and cure are concerned. The type of case to which I have just referred ordinarily is thought of as inoperable. Therefore the only means of treatment at our command is radiation.

It has long been a recognized fact that death of the laryngeal cartilages, particularly the thyroid cartilages, often follows radiation therapy (especially if the irradiation is given in dosage lethal to the cancer) with consequences fatal to the patient. Many explanations for this greater vulnerability of the laryngeal cartilages have been advanced but none seems entirely satisfactory. Many have come to consider this method of treatment as palliative rather than curative. Causes for greater vulnerability as advanced by various authors range from increased calcification[2] to invasion of the cartilage by cancer or by infection.[3] Zollner thinks invasion of the cartilage by cancer cells or by an inflammatory process renders it more susceptible to injury by radiation therapy. The literature, of course, abounds with reports and discussion of radiation injury to the cartilages but I have been unable to find any report of a detailed study of normal cartilage or cartilage which has been subjected to radiation.[4] An attempt at such studies is now being made in our laboratory in the Wash-

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ington University School of Medicine. This thought is well summarized by Salinger.[5] His bibliography is complete and the article also contains reports and direct quotations of opinions by radiologists who favor radiation over surgery and vice versa. I shall not go into these controversial questions in detail. Salinger does make special reference to Hautant's treatise, published in 1936, which "while stressing the question of radiosensitivity, also wisely gave due weight to consideration of the location and extent of the lesion."

Thomson[2] in his book says Hautant reported in October 1922 removal of the "anterior inferior calcified portion of the thyroid cartilage, preliminary to radiation" and in 1927 was able to report that three of six patients thus treated remained well. In so far as I have been able to discover from the literature, this is the only and the nearest approach to complete removal of the thyroid cartilages preliminary to radiation done prior to the time when I began doing it in 1940. It was thought then, and still is by many, that cancerous involvement of the cartilages was a contraindication to radiation therapy.[3, 5] Having had no experience wih radiation therapy with cartilages already involved, I, of course, had no opinion regarding that question. On the other hand I have seen at least two or three cases in which radiation therapy had resulted in death of the thyroid cartilages with the formation of abscess. In the first case, the patient was very sick (as they all are), in fact so sick that I was afraid to open his larynx. This patient improved with chemotherapy. He went home to a distant city for the week end even though he had considerable edema, and while at home choked to death. Since then I have seen two other patients, worse off than he was when they came in, in whom drainage with removal of dead cartilage was promptly instituted through a midline incision with extremely satisfactory results.

Harris and Klemperer[6] found that "the histologic picture of the biopsy did not fully conform to that of the entire tumor as regards the degree of differentiation and other cytologic features." I found the same to be true in a patient of Dr. Evarts Graham. This patient had an endobronchial tumor in which all four grades were observed when the tumor was studied as a whole; yet only one grade had been seen in the biopsy specimen. Harris and Klemperer state further that there is no material difference, as far as they can see, in the response to radiation by highly differentiated and less well differentiated types of cancer when the radiation is applied in lethal dosage. This has been my impression and I learned it early in my experiences with the small series of cases I am reporting here.

Coutard[7] calls attention to several differences between tumors of the cord and those in the subglottic region. He advises that we avoid surgery in treating cancer of the cord in favor of irradiation even though the cord is very mobile and even if the lesion is small. If the cord is fixed, surgery and not irradiation is indicated. He thinks the tumor of the subglottic region is easy to eradicate surgically. I hesitate, of course, to take issue with such an eminent authority, but I feel that where a cancer is still definitely localized to the true vocal cord, surgical removal is the best treatment and that limitation of movement is an indication of spread beyond the limits of the true cord with greater possibility of cure by irradiation after removal of the thyroid cartilages. It is possible that in the future we may come around to the point where all cancers of the larynx may be better treated by radiation therapy.

It has been said that some radiologists feel that the dosage of radiation necessarily must be reduced after removal of the thyroid cartilages, which places this operation at a disadvantage in their minds. We have had no trouble whatever with increased dosage and as a matter of fact it is our practice to give larger doses after removal of the cartilages and feel much safer about it than we were ever able to do before.

The plan of treatment in which I am interested at the moment, namely, surgical removal of the thyroid cartilages followed by radiation therapy, was begun by me in May 1940. It was thought of, and employed, at that time in the treatment of hopelessly late, inoperable cancers of the larynx and was done purely as a means of eliminating the danger of injury to the thyroid cartilages. The first group of patients were typical of those seen in the wards of city hospitals, usually old men with the types of constitutional disease usually seen in the aged, which alone placed them beyond the range of possible surgical intervention. In addition they were in the late stages of cancer with the usual degree of cachexia, anemia, and starvation.

The plan of procedure at first, and fairly closely followed up to the present, with certain refinements, was to use avertin anesthesia plus local anesthesia, and through a midline incision do a subperichondrial resection of the thyroid cartilages. Tracheotomy was done as the first stage, unless required as an emergency procedure before the operation for removal of the thyroid cartilages. After the wound closure a feeding tube was left in position. The patients withstood this operation satisfactorily. The wound healed by pri-

mary union and in eight to ten days, as a rule, they were ready for radiation therapy.

Before x-ray therapy was given every patient was warned that if they were to expect a cure they would of necessity receive burns and their mucous membrane would be irritated which would be terribly disagreeable.

If the radiologist is afraid of giving adequate dosage, which he frequently is, because of possible legal action on the part of the patient or his family afterwards, we feel this situation would be sufficiently covered by obtaining a signed statement, such as an operative permit which is required by many surgeons before operation is carried out. It will of course be understood that x-ray sickness sometimes is more severe in individuals with constitutional diseases, such as diabetes or paresis, and that treatment must occasionally be interrupted more than once because of extreme x-ray sickness. I have the feeling that some of these patients in whom x-ray treatment has been interrupted became radiation fast and when this occurs it is most difficult to secure response to radiation later.

Without professing to know or advance a theory why the thyroid cartilage is killed by radiation, we know it to be a fact, and unfortunately we cannot prophesy as to which one is more likely to be thus injured. In the meantime this plan of preradiation surgical removal of the thyroid cartilages is being carried out with very considerable satisfaction. Since my return to civilian practice in November 1944, eight cases of cancer of the larynx in varying stages, but none of which were confined to the true vocal cord, have been treated by this method. Of this group of patients, all except one are well and free of cancer, having had the thyroid cartilages removed and radiation therapy afterward. Before 1940, laryngectomy would have been recommended in every case. The cancers in this group of eight have not been as late or as widely distributed as were those in the group reported in the paper published in 1944,[9] nor were these patients as afflicted with senility or disease. Of the original group of 18 patients, 10 died of cancer and other causes as stated in the paper. There have been no operative deaths and of course no deaths from radiation therapy in the entire group. The group of eight patients are younger and healthier individuals and constitute much better risks, and the mortality rate has dropped off to an extremely low rate. The rate of curability and retention of function of the larynx is also much greater.

Some months ago one of my friends, a surgeon, questioned the statement that in the previous cases the glands in the neck had been

cancerous, saying that he could not find in the literature a single example of radiation cure of glands in the neck. In these early cases, the glands in the neck constituted a mass, firmly fixed, which was occasionally as big as one's fist and ranged from that size down to the size of a guinea's egg; it was so palpably malignant to me that I did not deem biopsy necessary. However, since this discussion I have routinely done a biopsy of the glands in the neck. In one case I could not find a gland but in the others I usually found a palpable gland the size of the rubber on a lead pencil which was not bound down by adhesions. This gland was the one in the carotid sheath at the jugulum. It was readily picked up and removed and invariably found to be either partially or totally invaded by cancer cells. In none of these cases could I feel glands elsewhere along the route between that point and the larynx. It seems conceivable to me that this gland may have been the first one in the neck to be involved and I suppose necessarily it was of microscopic size at first and therefore impalpable. This leads me to the belief that if laryngectomy is to be done, the surest guarantee of lasting cure is a bilateral neck dissection which should include the carotid sheath and all the glands between that point and the original lesion.

In the belief that radiation would destroy the cancer cells in all these lymphatic glands, as well as the original lesion, it has been our custom to refrain from any attempt at surgical removal of the tumor or the glands before radiation. We do insist on a positive biopsy finding before making a diagnosis of cancer. We insist upon telling these patients of the suffering they may expect as the result of radiation therapy before we start treatment. The tracheotomy tube is left in position until the reaction from radiation has thoroughly subsided and even then we are never in a hurry to remove the tube but we usually reduce the size to a No. 4 or 5 from a No. 7 or 8. A very helpful remedy in reducing the severity of skin burns is shaving cream.

The first case to be reported here was treated in May 1940. The patient had nearly lost his life by asphyxiation before tracheotomy. The terrors of this ordeal remain in his mind and he insists upon having a small tracheotomy tube left in his windpipe. This is a type of neurotic stenosis as described by Lynah[8] in 1918. With his tube (the lumen of which is covered by adhesive) in position the patient has a serviceable larynx with a husky voice which is due to the remaining fibrosis, often present after radiation therapy. The second case was treated in the fall of 1940. The patient has cicatricial stenosis of the larynx as the result of my having injured his

Fig. 1. Case 1.—Operation performed May 8, 1940. This photograph taken February 7, 1946.

Fig. 2. Case 2.—Operation performed October 8, 1940.

Fig. 3. Case 21.—Operation performed May 14, 1945; tube removed in January 1946. This photograph was taken February 7, 1946.

cricoid cartilage during the operation with subsequent collapse of this supporting ring. He has sufficient voice to enable him to earn his living as a salesman. The third case was that of a woman who had a cancer at the level of the cricopharyngeus muscle, with total obstruction of the esophagus by the cancer. This patient had radiation therapy after removal of the thyroid cartilages. She made a complete recovery from her cancer but her esophagus healed with a cicatricial web, so that when I returned from military duty and asked her to come to St. Louis for observation, I found she was still wearing a gastrostomy tube but was hale and hearty and had a good voice. The fourth patient treated during 1940 had a huge tumor of the epiglottis in the base of his tongue and larynx. He also had diabetes which was extremely difficult to control. He could not eat or breathe through the usual channels. X-ray therapy was of necessity interrupted several times because of x-ray sickness complicated by his diabetes. Finally his tumor failed to respond to irradiation and he died at the end of eight months.

Tables 1-3 give ages, dosage, results and other pertinent facts.

We realize that the time since these patients were first treated is all too short on which to base a claim of cure. We prefer to say that these patients either have succumbed to their disease or appear to be free of cancer and otherwise well after the period of time reported. This in itself is worthy of consideration when we realize that these derelicts who were in a hopeless condition with regard to treatment, have been well for the period of time stated and that the younger patients treated within the last two years, except for those still under treatment, have returned to their homes and their businesses. If such cases can be thus favorably influenced, it seems only natural to me to raise the question as to what may be accomplished by the application of this treatment in cases in which in the past we unquestionably would have recommended laryngectomy.

Until we began this method of treatment, we feared subsequent laryngeal stenosis as is suggested by Clerf:[1] "If the upper border of the thyroid cartilage is left intact there will be little tendency to development of stenosis of the larynx." There have been no cases of total stenosis. There has been one case of a high degree of stenosis due to loss of the cricoid cartilage (Case 2) but the patient still has a usable voice and earns his living as a salesman. One patient has a very husky voice due to intralaryngeal fibrosis which has persisted, but he also is able to earn a livelihood.

In summarizing, I have described a modified plan of approach in the treatment of inoperable cancer of the throat. This modification was devised in an effort to avoid radiation injury to the thyroid cartilages. We have demonstrated that all types of cancer will respond to radiation therapy. We have shown that the thyroid cartilages may be removed with very little risk to the life of the patient and without destroying the lumen of the larynx or interfering with laryngeal functions later. It is apparent to us that after subperichondrial resection a new framework for the voice box has been laid down within a few months.

In conclusion we feel that the surest way to avoid complications after radiation therapy is complete preliminary resection of the thyroid cartilages.

We believe it is necessary, in order to cure cancer, to give a larger dosage of radiation than is commonly used, even to the point of burns of the skin and extreme irritation of the mucous membrane. We are convinced better results may be obtained if these patients are carefully observed from day to day during their radiation period. There have been no deaths from radiation therapy.

We are aware that occasionally cancer is seen which from the beginning is radioresistant but fortunately these cases are very much in the minority.

We do not advance any explanation for the apparent lesser vulnerability of the laryngeal cartilages other than the thyroid cartilage nor do we have any opinion as to the probability of late changes due to radiation. Also we have no definite ideas, of course, as to the possibility of recurrence or continuation of the cancer in any given case. We can state definitely, however, that a number of patients with very late cancer of the larynx have been free of evidence of the disease for the period of time stated in the paper.

Since the longest period of time which has elapsed in any of our cases is six years, we cannot prophesy as to what may happen in the future with this plan of treatment, either as to permanent cure or late radiation reaction, but rather we are merely reporting results to this time of the 26 cases in this group.

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TABLE. 1.

CASE NO.	AGE	CLINICAL AND MICROSCOPIC DIAGNOSIS	DATE OF REMOVAL OF THYROID CARTILAGES
19. L. D.	56	Epidermoid Carcinoma	2-26-45
20. H. S.	54	Squamous Cell Carcinoma, well-differentiated, Grade I	11-22-44
21. J. Z.	45	Squamous Cell Carcinoma, Grade III	5-14-45
22. M. K.	52	Epidermoid Carcinoma, Grade II	1-24-46
23. T. M.	60	Carcinoma of larynx. Metastasis to lymph nodes and perichondrium. No grading.	1-14-46
24. G. M.	53	Epidermoid Carcinoma, Grade III	9-4-45
25. C. L.	70	Epidermoid Carcinoma, Grade III	8-20-45
26. B. Z.	64	Epidermoid Carcinoma	3-21-46

Cases 1 to 18 reported in paper: Treatment of Inoperable Cancer of the Throat.[9]

TABLE 2.

CASE NO.	DATE RADIATION STARTED	DATE RADIATION CONCLUDED	DATE AND RESULTS OF LAST FOLLOW-UP EXAMINATION
19. L. D.	Due to complication of Vincent's angina, did not start until 3-26-45 and treatment was interrupted several times	6-14-45	Died August 22, 1945.
20. H. S.	12-4-44	12-21-44	Living and well and carrying on his business. No evidence of cancer 2-7-46 and a report from his physician two weeks ago was that at this time there is no evidence of cancer.
21. J. Z.	5-23-45	6-16-45	Feeling fine and working every day. Tracheotomy tube was removed 1-8-46. No evidence of cancer 6-6-46.
22. M. K.	7-27-45 1-28-46	8-3-45 3-23-46	Feeling well and managing his business. Tube removed 4-21-46 and no evidence of cancer.
23. T. M.	1-24-46	2-15-46	Tube removed 5-6-46. No evidence of cancer 6-11-46.
24. G. M.	Still under treatment		
25. C. L.	11-18-45	12-7-45	No evidence of cancer 2-7-46. Feeling fine, gaining weight and working daily.
26. B. Z.	4-1-46	5-3-46	6-14-46 patient reports feeling fine. No sign of cancer.

Cases 1 to 18 reported in paper: Treatment of Inoperable Cancer of the Throat.[9]

TABLE 3.

AMOUNT OF RADIATION GIVEN

Case No. 19.	L. D.	1600 R. U. to each side of neck but due to complications radiation was interrupted several times. St. Mary's Hospital, East St. Louis, Illinois.
Case No. 20.	H. S.	2200 R. U. to each side of neck, December 4, 1944, to December 21, 1944, inclusive. Barnes Hospital, St. Louis, Missouri.
Case No. 21.	J. Z.	2600 R. U. to each side of neck, May 23, 1945, to June 16, 1945, inclusive. Barnes Hospital.
Case No. 22.	M. K.	1400 R. U. to each side of neck, July 27, 1945, to August 3, 1945, inclusive. 2200 R. U. from January 28, 1946, to March 23, 1946, inclusive. Barnes Hospital.
Case No. 23.	T. M.	2600 R. U. to each side of neck, Januay 24, 1946, to February 15, 1946, inclusive. Barnes Hospital.
Case No. 24.	G. M.	Still under treatment.
Case No. 25.	C. L.	1800 R. U. to each side of neck. St. Mary's Hospital, Decatur, Illinois.
Case No. 26.	B. Z.	2400 R. U. to each side of neck, April 1, 1946, to May 3, 1946, inclusive. Barnes Hospital.

Cases 1 to 18 reported in paper: Treatment of Inoperable Cancer of the Throat.[9]

LXIV

CONGENITAL CYST OF THE MEDIASTINUM PRODUCING COMPRESSION OF THE LOWER TRACHEA AND ESOPHAGUS

GABRIEL TUCKER, M.D.

PHILADELPHIA, PA.

A male infant, three months of age, was admitted to the Graduate Hospital on December 22, 1945, because of increasing respiratory difficulty. At birth, which was a breech delivery with forceps, the child's color was good but noisy respiration was noted. A heart murmur also had been observed at birth by the pediatrist, but this was reported to have disappeared. Deformity of the feet, bilateral equinovarus, was also noted. The respiratory difficulties had gradually increased and at the time of admission were continuous but did not produce cyanosis. The child had gained in weight and his appetite was good. All efforts of the pediatrist to relieve the infant's respiratory difficulties had been unavailing.

On admission the child seemed well nourished and his temperature was normal. Difficult breathing was the outstanding symptom, the dyspnea being of the expiratory type, as indicated by prolonged expiration and the barrel-shaped chest.

Roentgen-ray examination showed the neck to be negative except for anomalous development of the cervicodorsal vertebrae. This resulted in slight deformity of the upper thorax. The esophagus was deviated considerably toward the right posteriorly, the trachea toward the right anteriorly. The trachea also showed marked conpression by a mass lying between the esophagus and the trachea. Because of the other congenital anomalies which the child showed it was suggested that this was also a congenital anomaly, most probably of the great vessels. Neoplasm of congenital origin, such as dermoid or teratoma, was considered as a possibility.

The thoracic surgeon in consultation agreed that a tracheotomy should be done in an attempt to relieve the dyspnea so that surgical removal of the mass might be attempted. Before tracheotomy a 4 mm. bronchoscope was introduced showing a normal larynx and upper trachea. Compression of the trachea by pressure on its poster-

ior wall was noted, producing expiratory collapse. This compression extended downward to the level of the bifurcation. Tracheotomy relieved the dyspnea temporarily. The child continued to take nourishment satisfactorily for several days but the dyspnea increased and remained the same type. The mass seemed to be growing larger. Pulmonary complications developed despite meticulous after-care following the tracheotomy and the administration of penicillin, and the child died one week after admission from pulmonary complications with cardiac failure.

Postmortem examination showed a mediastinal cyst, probably rising from the trachea or the upper gastro-intestinal tract. The trachea and the left main bronchus were compressed by this mediastinal cyst. The esophagus was displaced posteriorly; a patent ductus anteriosis was also noted.

Microscopic examination of a portion of the wall of the cyst showed it to be lined with simple columnar epithelium. The capsule was made up of fibrous tissue with some smooth muscle fibers and several sections of stratified muscle bordering on the fibrous capsule. The cyst was filled with pink-staining pseudomucinous material. The cyst probably originated from the dorsal aspect of the trachea to which it was closely applied. No direct connection to either the trachea or the esophagus could be demonstrated.

250 South 18th Street.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 7, 1946

THE PRESIDENT, DR. JOHN F. DELPH, IN THE CHAIR

Pulmonary Complications In Otolaryngology

SURGICAL ASPECTS

WILLARD VAN HAZEL, M.D.

(Abstract)

Frequently patients are seen after a diagnosis has been made or after conservative measures have failed to correct the existing complications. With regard to the embolic versus the aspiration theory of the etiology of lung abscess I am confident that both causes may be present. It has been shown in experiments on dogs that pus from an abscess in the human being, introduced into the bronchial tree in the dog, did not produce an abscess unless the pus were mixed with blood, when the incidence was increased greatly. Therefore this condition has significance from the viewpoint of prophylaxis, and if abscess of the lung can be prevented, the patient has been spared a serious complication. Bleeding should be controlled in so far as it is possible, and if it does occur, the blood should be sucked out. If the patient is under general anesthesia, the head should be turned to the side or the patient should be placed on his abdomen; every precaution should be taken to prevent aspiration of blood. When a tonsillectomy is performed under general anesthesia there is always blood in the bronchial tree. An embolic process would suck blood into the blood stream and it should be filtered out through the lung. On the other hand, blood that clots in a small bronchus may be the cause of an embolic process, just as any

other foreign body. This is emphasized because in most instances it can be prevented. Men who perform tonsillectomies are usually general surgeons in small communities, and if they can be shown that prophylaxis may spare this complication, a service is rendered to the medical profession and to the patients.

Improved x-ray technic, improvements in anesthesia, have all been valuable, but a careful physical and laboratory study is essential. Differential diagnosis is still difficult in many cases and interpretation of x-ray films by the physician himself, not by a laboratory technician, is frequently of great help.

Penicillin and some of the sulfonamides have been a great boon in dealing with suppurative processes in the chest. However, the old and tried remedies should not be disregarded. Treatment must be varied to suit the individual case and when conservative measures fail, surgery may be necessary to achieve the desired result.

Improvement in the abscess may give a sense of false security; the symptoms may recur with blockage or respiratory infection. The cavity must be eliminated. If the abscess is the type known as acute putrid abscess, which is rapidly advancing and usually has a very thin wall, surgery is necessary and should not be delayed. The effectiveness of pneumothorax has long been known but at times is used with disastrous results.

A central abscess will usually clear up spontaneously. Peripheral abscesses, with small bronchioles entering them, with poor drainage and edema, require surgical drainage or lobectomy. Not all patients with bronchiectasis need lobectomy. However, when colds and fever occur, when the patient is confined to bed for a much longer period than is usually necessary, that is an indication for careful observation.

These abscesses should be prevented by any means possible. If they fail to respond to conservative measures, certain surgical procedures may be effective. This holds true also with bronchiectasis. Our present treatment is safe and corrective, whereas treatment in the past has been at best only temporary, with little hope of prolonging the life span; if the condition is present before the age of ten, only 10 per cent have lived beyond the age of 40. By earlier diagnosis and early treatment this picture may be changed.

MEDICAL ASPECTS

ROBERT KEETON, M.D.

(Author's abstract)

I. *Foci of Infection*: (a) Blood stream dissemination of bacteria following eradication of foci of infection.

It is usually desirable and frequently imperative to eradicate foci of infection. Formerly no specific precautions were taken when these were removed. More recently protective agents have been made available for this purpose. Their effectiveness obligates their use.

The dentists have shown that a bacteremia follows the extraction of an infected tooth. This lasts from 12 to 24 hours. If one cultures the blood of patients with acute infections of the upper respiratory tract shortly after onset, alpha and beta streptococci will be recovered. When the patient has had time to mobilize his antibodies, these organisms disappear. However, they may frequently be recovered from the urine, indicating that they were trapped by the kidney and filtered out by the glomeruli. The same blood stream dissemination follows tonsillectomy and other operative procedures of the nose and throat.

(b) Complications following eradication of foci of infection: The complications following bacterial dissemination may now be listed.

Lung abscess: The otolaryngologists are not agreed as to the mechanism of production of lung abscess, but they are agreed that it commonly follows tonsillectomy. It is also clear that it may arise from a pulmonary embolus with an infected clot.

Bacterial endocarditis: Bacterial endocarditis is a complication that follows the dissemination of streptococcus viridans. The organisms become implanted on a valve previously damaged by rheumatic heart disease. The possibility of this complication should be considered when a tonsillectomy is proposed to such patients.

Exacerbation of latent nephritis: It is becoming more generally accepted that nephritis results from the antigen-antibody reactions which occur within the kidney. In latent nephritis the renal cells have developed a high degree of local immunity to antigens formed in a previous attack of sore throat, scarlet fever, or other similar diseases. If the tonsils continue to harbor these organisms,

the antigen in much reduced doses will continue to reach the kidney cells and keep them in a reactive state. Hence the nephritic reaction never quite subsides and the patient does not completely recover. If a tonsillectomy is performed, there is a larger dose of antigen liberated and an exacerbation of the nephritis occurs. This is commonly spoken of as an exacerbation of a chronic nephritis.

(c) Prevention of bacterial dissemination: The prevention of bacterial dissemination following the eradication of chronic foci of infection may be accomplished by administration of sulfadiazine or penicillin. If sulfadiazine is to be used, it should be started the day preceding the operation and given in doses sufficient to establish a therapeutic level on the morning of the operation. If penicillin is to be used, it should be started on the morning of the operation. Due to the variable sensitivity of bacteria to penicillin, a sufficiently high level must be maintained to cover all organisms. Doses of 50,000 units should be given every one and a half hours for the first twelve hours, and every three hours thereafter.

II. *Chronically Congested Nasal Mucosa.* The etiology of this condition is best known to the otolaryngologist. In some cases there is a frank bacterial infection of the sinuses. In others there is an element of allergy present. It is not always clear as to whether the antigen is of local origin and due to the bacteria, or extraneous to the nose and due to environmental factors. The general effects on the patient are the same irrespective of the etiology. Thus if the antigen comes from the environment, the congestion traps the more or less normal bacteria within the nasal mucosa, and their products or the bacteria themselves become disseminated. If there is a chronic sinusitis, the associated congestion and the poor drainage facilitates the dissemination of the bacteria.

The internist sees two common clinical complications, chronic asthmatic bronchitis and benign albuminuria. He has little to offer in the treatment of chronic bronchitis. He feels that treatment is best directed toward the nose. In the treatment of benign albuminuria some progress has been made. If one studies these patients he will find that the blood pressure is normal, the kidney function is unimpaired, and that albuminuria is absent when the patient is lying flat in bed. As soon as he gets up and around, rather sizable amounts of albumin appear in the urine. Only occasionally are red blood cells or casts found. The condition has sometimes been spoken of as "orthostatic albuminuria." These patients are improved by measures which unblock the nose, including transfer of the patient to a dry warm climate. In addition to this the albumin becomes

less under small daily doses of histamine given subcutaneously. This treatment is based on the assumption that the albuminuria is a response to a low concentration of antibodies in the kidney. The antigen which produced these antibodies originated in the congested nose. The condition is the same as that seen in nephritis, but it is milder because the local renal cell immunity is low. Frequently repeated small doses of histamine tend to abolish the renal cell immunity. When this is abolished, the antigen from the nose is ineffective in producing a renal response and the albuminuria disappears. The patient should be taught to inject himself with the histamine, and he must be willing to continue the injections for a long period (one year or more).

III. *The Congested Lung.* The circulation through the lung becomes slowed and passive congestion develops under a wide variety of conditions which concern the otolaryngologist and the internist. Among these are congestive heart failure, pneumonitis secondary to bronchitis, atelectasis, pulmonary embolism, and hypostasis secondary to cerebral arterial accidents and abdominal operations. If pathogenic organisms can be prevented from growing in the congested lungs, the patients usually recover. If this precaution is neglected, death follows. Consequently the prophylactic administration of penicillin is required in all situations which may be associated with pulmonary congestion.

DISCUSSION

DR WALTER H. THEOBALD: I am sure we have all appreciated this excellent presentation. Dr. Andrews touched upon anesthesia; Dr. Van Hazel pointed out the importance of prophylaxis but did not tell us what to do. My mind went back to my interne days, when a mask something like an opera hat was put over a child's face, an ether can was emptied into it; the tonsils and adenoids were removed and the patient sent back to his bed and often allowed to bleed until it stopped. It is amazing that more complications such as lung abscesses did not occur.

Today, however, one of the best departments in our hospitals is the department of anesthesia. One can obtain any type he chooses. I would like to point out the advantages of intratracheal anesthesia which, to my mind, is ideal. One has profound and deep narcosis, while the intratracheal tube acts as a stopper. At the completion of the operation a smaller catheter introduced through the tube aspirates any blood that remains in the trachea, and it is a relief to know that

the trachea is clear. Also it is important to be sure that when the patient leaves the operating table there is no bleeding; that the tonsil area is dry and the adenoid area is dry. Often bleeding may occur after the patient is returned to his room, and we do not know about it until considerable bleeding has taken place. These two points, I believe, are important in prophylaxis.

DR. G. H. MUNDT: In considering general anesthesia for tonsillectomy, we should also bear in mind the advisability of local anesthesia, not desensitizing the pharynx more than necessary; also we should consider the larynx and the trachea. It used to be routine to spray a five per cent solution of cocaine hydrochloride into the pharynx prior to the injection for tonsillectomy. I have never done that, nor can I understand why this procedure is continued. If the patient is brought to the operating room on a cart, operated on in the upright position, the injection for local anesthesia can be done comfortably; I think it reduces the danger of inhalation of material into the larynx and into the trachea.

Meeting of Monday, February 4, 1946

THE PRESIDENT, DR. JOHN F. DELPH, IN THE CHAIR

The Normal and the Pathologic Structure of the Fissular Region of The Temporal Bone*

BARRY J. ANSON, Ph.D.

AND

EARL W. CAULDWELL, M.D.

(Author's abstract)

The auditory apparatus is remarkable not only for the intricate complexity of its nervous elements, but also for the exceptional constitution of the bone in which its sensory organs are housed.

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*From the Departments of Anatomy and Otolaryngology, Northwestern University Medical School.

Based upon a study of more than 200 serially sectioned human temporal bones (from the stage of the 10 mm. embryo to that of the 80 year old adult); many of the series were studied through the courtesy of Dr. Theodore H. Bast, of the University of Wisconsin.

The adult temporal bone is "petrous" only where it serves as a bed for membranous canals and vesicles; the petrous part is actually only the cochlear, vestibular and fenestral portions; marrow and air spaces occupy much of the remainder. In attaining such structure the capsule passes through a complex succession of developmental stages. In the course of this transformation it comes to contain a fissure of perilymphatic nature, termed the "fissula ante fenestram," named for its position anterior to the fenestra ovalis.

This fibrous tract begins externally (i.e., on the middle ear side) on the medial wall of the tympanic cavity. It ends internally (labyrinthic side) at the junction of the vestibule with the scala vestibuli. Although typically the fissular tract is filled with a moderately vascular fibrous tissue, it is sometimes occupied by otosclerotic bone; but more frequently, in the young at least, by a large mass of cartilage. For this reason the morphogenesis of the fissular region is worthy of detailed study.

The fissula develops as an anteriorly directed outpushing of the periotic connective tissue of the vestibule in the 50-mm. embryo. Around the projection the precartilage of the primitive capsule breaks down—in just the way that the same tissue is removed to permit any part of the labyrinth to enlarge.

The fissula gradually widens and lengthens, reaching the opposite, or tympanic, surface of the capsule in the 100-mm. embryo. In the 111-mm. fetus the fissula is histologically definite, through contrast between its content and the surrounding cartilage.

During the next month of fetal life the fissula attains maximum size. Concurrently, the cochlea, vestibule, canals, otic capsule, stapes also reach full, adult dimensions—when the fetus is in the middle of its intra-uterine life. The remaining prenatal months, and all the years of infancy, childhood and manhood are available, so to speak, for histologic maturation or for the genesis of pathologic tissue.

At this stage—midterm fetus—cartilage of the primordial capsule remains as a tube within which is lodged the fibrous tissue derived from the original periotic extension. This cartilage is continuous with a chondral rim bordering the oval window. This continuity is maintained even in advanced age. At the tympanic extremity of the fissula there is similar continuity—later to be interrupted. Perichondral bone now covers the capsule. Between this plate-like bone on the periphery and the cartilage enclosing the fissula, at the core of the capsule, osseous tissue of an unusual type is being formed

through rapid conversion of cartilage into intrachondral bone. This feature will be considered later.

The cartilage enclosing the fissula has been almost completely removed by the 210-mm stage. It is represented only by a thin layer of perichondrium. Bone has spread to form an uninterrupted wall for the fissula. The fibrous content remains unchanged. The marrow space contains almost no bony spicules.

In the otic capsule of the infant the extensive marrow spaces of the fetus have been largely obliterated by surprisingly rapid production of endochondral bone. Cartilage, produced by the perichondrium present in the preceding stage, now immediately surrounds the connective tissue of the fissula. Once formed this cartilage remains throughout life in all normal cases. At the tympanic orifice cartilage is wanting. Continuity with that of the oval window has been lost by intervening growth of perichondral bone.

So, when once the fissula is formed, as a fibrous stripe in cartilage, the established relationship between these two tissues is maintained despite profound rebuilding of the surrounding capsule. As will be pointed out, this cartilage is the fundamental tissue in the pathologic changes basic to otosclerosis.

Typically, the fissula traverses the capsule from tympanic orifice to vestibular opening. Its form is that of a flattened passage—narrow, tall and C-shaped. However, in some specimens a third orifice is present, opening into the oval window (i.e., fenestral orifice). Occasionally the three are continuous.

In such instances (126 mm. embryo) the fissula appears to be a deep cleft in the capsular wall. It extends without interruption from the wall of the vestibule upward and outward, across the rim of the oval window to the middle ear. Now, were the cartilage lining the fissula to undergo neoplastic growth, its new tissue could invade the oval window. Here, then, is the route for invasion ready-made. To cause deforming pathologic changes there is required only chondromatous growth or growth followed by replacement with sclerotic bone. Such growth seems likely to occur when fissulae are oversized. When large it is likely to extend, as a cupola, above the level of the tympanic orifice. Histologic activity seems to be invited by space of this kind; cartilage, produced by the persistent perichondrium, comes to occupy part, or obliterate all, of the intra-osseous defect. These chondromatous nodules will now be discussed.

In some specimens, and especially in the late fetus and the infant, the cartilage lining the fissular channel does *not* remain quiescent. For example, in a 180-mm. (midterm) fetus a precociously developed mass of immature cartilage has displaced the normal fibrous tissue of the fissula. It appears as a chondromatous nodule. It extends above the level of the tympanic orifice, fills the space at that opening and at the fenestral opening, and it reaches the vestibular orifice. A chondroma of this kind may be limited to the cupola of an oversized fissula. It may fill the entire space. It may be associated with sclerotic bone. We believe that this association is significant and that the chondroma is invaded by vascular bone to produce the nodular mass encountered in otosclerosis. In advanced stages the otosclerotic bone reaches the oval window. This bone is highly vascular, and somewhat invasive.

To summarize:

(a) The otic capsule in the temporal bone of man has passed through a remarkable series of developmental changes in attaining its adult form.

(b) In the antefenestral region of the capsule, between the oval window and the cochlea, the structure is exceptional in that it contains a large number of cartilage islands, or islets of intrachondral bone, and is crossed by a channel (termed the fissula ante fenstram) which normally is occupied by connective tissue and bordered—at least at its vestibular end—by a thin layer of cartilage.

(c) While typically the fissula is a narrow, C-shaped channel, occasionally (and most frequently in the young) the fissula is enlarged and contains a cartilage mass, or chondroma, produced by abnormal growth of the cartilage lining the fissula.

(d) In rare cases, sclerotic bone, developing rapidly, in turn replaces cartilage, to obliterate part or all of the original fissular space, sometimes spreading even to the oval window and impinging upon the stapes.

(e) Fundamentally, then, the formation of otosclerotic bone is an almost natural consequence of congenital failure in bone development in a specialized area of the otic capsule.

DISCUSSION

DR. E. W. HAGENS: We are indebted to Dr. Anson for his excellent presentation of this interesting portion of the petrous

bone. He and his co-workers, Drs. Wilson and Bast and others, have been working on this and on other phases of the temporal bone for many years. In his discussion he does not consider the etiology of otosclerosis, but emphasizes that in the development of the petrosa the fissula ante fenestram has potentialities that in some instances may lead to the formation of a chondroma in late fetal life which, in turn, can be changed into an otosclerotic type of bone.

In a series of temporal bones from 12 children, aged 13 months to 14 years, I found seven children showing a cartilage focus in the "site of predilection." The five children not showing cartilage foci were the older ones, aged 5 to 14 years.

Dr. Anson finds that the narrower part of the fissula, containing loose connective tissue and located near the vestibule, is the last portion of the fissula to be involved when cartilage or otosclerotic bone overgrows this region. Bast has presented slides of a case of otosclerosis where this part of the fissular space is still present, although surrounded by otosclerotic bone. It is apparent that in the evolution of the otosclerotic bone the process begins in the chondroma tissue and not in the fissular space. My own sections have been so far advanced that only the niche of the fissula at the margin of the vestibule can be seen, all the remaining tissue being otosclerotic bone.

In addition to occurring at the "site of predilection" otosclerosis is found in other places, such as around the cochlea, adjacent to the semicircular canals, at the round window, and rarely in other areas. Otosclerotic foci in these additional sites occur infrequently compared to that in the region of the fissula ante fenestram, but inasmuch as they are found, it is interesting to speculate whether the changes described by Dr. Anson may have occurred in these areas of the petrosa of such cases. Another point of interest concerns the fossula post fenestram. Drs. Anson and Bast have found the fossula to be similar to the fissula ante fenestram, excepting that it occurs less regularly, is more often incomplete, and that chondroma and bone formation occur in only five per cent of the cases revealing a fossula. Therefore, while otosclerosis would naturally not be expected to occur in the fossula region nearly so often, it is strange that it does not seem to be found there at all.

In the petrous bones of younger children and in the newborn in particular, foci of cartilage are found in areas other than at the fissular region. These foci, however, are usually in connection with the posterior part of the petrosa near the semicircular canals and

are no doubt areas that have not as yet completed their cycle into bone. Bast has emphasized the need for such a situation in order for full development of the canals to take place. Otosclerosis has occasionally been found in these areas, yet these cartilage foci probably do not have the same significance as the cartilage formation at the "site of predilection" reported by Dr. Anson.

DR. GEORGE E. SHAMBAUGH, JR.: I would like to ask Dr. Anson what he would say to Dr. Guild's objection to his theory. Guild said he has seen numerous foci near the region of predilection, but not actually in contact with the fissula; therefore he did not think the fissula had anything to do with the pathogenesis of otosclerosis.

DR. H. B. PERLMAN: I would like to have an expression from Dr. Anson as to the function of the fissula ante fenestram. This structure, so far as I know, is peculiar to man and is not seen even in the otic capsule of the monkey.

DR. BARRY J. ANSON (closing): In reply to Dr. Shambaugh, I should not have inferred that this is the only histologic possibility. However, in the fissular region specifically, the histologic association and succession do seem to us to be significant.

In reply to Dr. Perlman, I have no settled opinion as to what function the fissula serves. It was suggested many years ago that it might serve as a synchondrosis for capsular expansion. We believe there is no developmental basis for that notion. It is not in the proper place; it is too small; it persists long after the time it could be of any such service; and it appears prior to the stage at which it would be essential were it acting in that capacity.

Factors Influencing Osteogenesis Following the Fenestration Operation: Observations On the Monkey

GEORGE E. SHAMBAUGH, JR., M.D.

(Author's abstract)

The history of the fenestration operation has been the story of the search for a method of making a labyrinthine fistula that would remain open. Experimental study of the fenestration operation on the monkey has been carried out during the past four years at

Northwestern University. The same technic which has been used successfully on the human, namely, the nov-ovalis operation with constant irrigation and observation with a microscope, was used on the monkeys. Various modifications of this technic were also used. Histologic study of these monkeys at various intervals after the operation has revealed the following factors that influence osteogenesis after the fenestration operation:

1. The sluggish responses to trauma of the enchondral layer of the labyrinthine capsule.

2. The stimulating effect on osteogenesis of bone dust or chips.

3. The inhibiting effect of stratified squamous epithelium on osteogenesis.

4. The stimulating effect on osteogenesis of trauma to the endosteum.

5. The inhibiting effect on osteogenesis of periosteum.

6. The inhibiting effect on osteogenesis of a smooth polished bone surface.

7. The stimulating effect on osteogenesis of fibrosis within the perilymph space.

8. The effect of a close adhesion between the skin flap and the endolymphatic labyrinth in keeping the mouth of the fistula open.

The application of these factors brought out by the monkey experiments to the human operation have resulted in a technic by which the incidence of bone closure has been reduced to less than five per cent two years after the operation. At the present time, postoperative labyrinthitis constitutes a more important cause for failure in the fenestration operation than does bone closure.

DISCUSSION

DR. H. B. PERLMAN: For several years we have been interested in the repair processes of the labyrinth in response to injury. There are so many factors involved that it is difficult to devise experiments where a single factor can be studied. The histologic picture is difficult to evaluate and permits a variety of interpretations. Of course, the great interest is in the problem of arresting osteogenesis because of the importance of a wide open fistula for functional improve-

ment in the fenestration operation. The monkey labyrinth and middle ear closely resemble that of man and naturally lend themselves for study of the histologic changes following this operation. Dr. Shambaugh has demonstrated, and Dr. Lindsay and I can confirm, that even though the structures are much smaller than in man, it is possible to do a fenestration operation on the monkey with the same attention to meticulous detail as one does the operation in man. One difficulty that may be pointed out is that in the postoperative period infection is more likely to occur in the monkey.

Dr. Lindsay has operated on a large series of animals and has been successful in keeping the fistula open for as long as 14 months. Thiersch grafts and conjunctival grafts have been tried in addition to the conventional posterior meatal flap. It is his impression that the important factor for insuring an open fistula is the ability to obtain uninterrupted contact between the membranous canal and the covering flap. It is probably the union of these two that prevents the osteogenic tissue from growing between them and thereby closing the fistula. In order to obtain intimate apposition of these structures it is necessary to prepare the cavity so that the fistula and the membranous canal will be at the top of a mound. Once apposition is made, it must be maintained by packing for a sufficient period to insure union. The role played by the various elements (endosteum, periosteum, bone chips, blood, epithelial flap, enchondral layer) is open to different interpretations. I do not think I would agree with Dr. Shambaugh that blood in itself is an important factor in promoting bone formation in the labyrinth at the site of the fistula. I have seen blood cells in the perilymph of the cochlea of animals long after operation without any evidence of local reaction. However, as experimental and clinical experience indicates, the present degree of success in keeping the fenestrum open is dependent in great measure upon a meticulous preparation of the tympanomeatal flap, of the mastoid and of the fistula, using irrigation for the latter. The preparation of the fistula should be carried out with a fine drill under sufficient magnification (binocular scope) so that the membranous canal can be seen clearly and the perilymph cleaned of all bone particles. The final appearance of the fistula before it is covered with the flap is a clean wide defect with the membranous canal floating in clean perilymph.

Dr. George E. Shambaugh, Jr. (closing): The reaction of the arachnoid mesh at the fistula is very striking. It eventually disappears, but in some cases the fibrosis might persist. This reaction is, I believe, due to hemorrhage. We have found that blood may

remain unchanged in the cochlea for as long as four weeks. At the fistula we never found red cells in the perilymph space longer than one week after operation. It is apparently not infection that causes the reaction, but blood.

As to the relative evaluation of these factors that influence osteogenesis, it is difficult to judge. In different ears these various factors operate to different degrees. In one ear there will be profuse growth of endosteal bone which causes closure, in another bone dust. I think all these factors that influence closure should be taken into account, so as to get a maximum number of cases that will remain open.

Abstracts of Current Articles

EAR

Tympanosympathectomy: A Surgical Technic for Relief of Tinnitus Aurium.

Lempert, Julius: Arch. Ofolaryng. 43:199-212 (March) 1946.

The author advances the theory that tinnitus in some individuals is caused by diseased sympathetic ganglion cells found in the tympanic plexus. He bases this theory on observations indicating that middle ear disease is accompanied by tinnitus, unless the disease process destroys the tympanic plexus, in which case there is no tinnitus.

The operation is done by lifting the lower half of the drum from its frame, thus exposing the middle ear cavity. The mucous membrane covering the promontory together with the branches of the contained nerve plexus is removed. The drum is then replaced.

This operation can be done without disturbing the hearing. Of 15 patients thus operated, 10 were completely freed of their tinnitus.

Only patients complaining of very severe tinnitus have thus far been operated on.

HILDING.

The Reaction of Tullio in Resonance, Etc.

Van Eunen, A. J. H., Huizinga, H. C., and Huizinga, E.: Acta Oto-laryngologica 30:265, 1943.

An exhaustive study employing the reaction of Tullio in pigeons is considered to support the Helmholtz theory of hearing. The authors ascribe the following functions to the middle ear: transmission of sound, sensitiveness for given frequencies, protection of the internal ear and generation of subjective tones. Under normal conditions sound does not reach the cochlea through the sound windows.

HILL.

Otitis Externa.

Syverton, J. T., Hess, W. R., and Krafchuk, John: *Arch. Otolaryng* 43:213-225 (March) 1946.

The authors studied 50 men presenting otitis externa in 72 ears. Cultures of all of the ears were made and the bacterial and fungus flora studied. Cultures were also made of the flora in 16 normal ears. The latter acted as controls.

The most striking result of the study was that only 25 per cent of the ears yielded fungi, and these were not especially pathogenic.

The microbiologic flora in otitis externa differed from the normal in the same clime by the inclusion of one or more pathogens. Among these were seudomonas aeruginosa, Ozaenae, Staphylococcus aureus hemolyticus, Aspergillus tropicalis, Actinomyces proteus.

HILDING.

A Contribution to the Diagnosis of Congenital Deafness.

Ojala, Leo: *Acta Oto-laryngologica* 31:127, 1943.

The author feels there is a definite causal relation between albinism and congenital deafness. Experimental studies on an albinic congenitally deaf cat suggest the theory that pigment is necessary, possibly as a catalyzer in certain secretory functions, and its lack may result in degenerative phenomena.

HILL.

PHARYNX

The Aspiration Risk in Tonsillectomy under Narcosis.

Bergovist, B.: *Acta Oto-laryngologica* 31:152, 1943.

The aspiration risk in tonsillectomy was carefully studied in a large series of cases. The author feels that aspiration during actual operation can be avoided by careful attention to technique. The critical period is during the recovery from anesthesia, postoperative bleeding being the most usual cause.

HILL.

Transitory Occurrence of Horner's Syndrome after Paratonsillar Anesthetization for Tonsillectomy.

Hold, E. H., and Godtfredsen, E.: Acta Oto-laryngologica 30:156, 1942.

Fifty per cent of 22 patients who were given injections of an anesthetic agent before tonsillectomy presented transitory Horner's syndrome, showing that the sympathetic fibers to the eye are often affected, despite careful technique.

HILL.

BRONCHI

On Benign Bronchial Tumors.

Leegaard, T.: Acta Oto-laryngologica 30:383, 1942.

The author considers that the majority of benign bronchial tumors such as adenomata and cylindromata histologically are variably differentiated mucous and salivary gland tumors. Treatment depends upon radiosensitivity; electrocoagulation or roentgen therapy or a combination of both should be employed.

HILL.

MISCELLANEOUS

On the Problem of Focal Infection.

Diamant, M.: Acta Oto-laryngologica, Trans. IX Nordic Congress for Oto-laryngology, Supplement, 1939.

The author considers that changes in both foci and peripheral morbid processes as to bacterial content are parallel phenomena and that the idea that the so-called foci represent the etiological factor of the peripheral lesions is incorrect.

HILL.

The Oral Lesions of Monocytic Leukemia.

McCarthy, F. P., and Karcher, P. H.: New England J. Med. vol. 234, No. 24 (June 13) 1946.

Oral lesions represent a frequent and early finding in the various forms of leukemia. This is especially so in monocytic leukemia, except in endentulous mouths. The most frequent signs are bleeding from the gums, swelling, necrosis and ulceration of the gingivae.

In all such cases which may be considered as Vincent's infection careful examination of the blood is essential to rule out severe blood dyscrasia.

HILL.

A Study of the Attitudinal Reflexes of Magnus and De Kleijn in Thalamic Men.

O'Neill, Hugh: Arch. Otolaryng. 43:243-282 (March) 1946.

This is an extensive study of a 40-year-old woman who was injured in an automobile accident on September 23, 1937, suffering severe brain injury. She was operated on February 7, 1938, at which time it was found that the brain had been torn and lacerated on the left side and that there was a huge fluid subdural hematoma on the right. She suffered a cone compression of her temporal and parietal lobes, and by way of the incisura both the cerebellum and the brain stem were squeezed, with gradual release of the bulbopontile mechanism. The patient exhibited decerebrate rigidity and passed through the various phases of the thalamic, midbrain, and brain stem animal. This clinical state was anamnestically present for four months. For several days there were displayed Walshe's criteria: double pyramidal system spasticities with complete double release, the bilateral presence of the reflexes of Magnus and de Kleijn and hypothermia. The patient recovered after operation. The article is well illustrated with photographs of the patient in various reflex states.

HILDING.

On the Occurrence of Eosinophilia in Blood Tissue and Secretion in Certain Rhinal Affections.

Anderson, H. C.: Acta Oto-laryngologica, Trans. IX, Nordic Congress for Oto-laryngology, Supplement, 1939.

In contradistinction to blood or nasal secretion eosinophilia, which only exceptionally fails to be detected in allergic patients, tissue eosinophilia is of little diagnostic value.

HILL.

Otogenous Cerebral Abscesses.

Juul, A.: Acta Oto-laryngologica 30:192, 1942.

The author makes a careful study of 41 otogenic brain abscesses. The mortality was about 66 per cent. Increased cells in the spinal fluid with normal or near normal temperature should make one suspicious of brain abscess. In most cases early operation is advocated;

the closed drainage method of LeMaitre is preferred. In cases of inefficient drainage, open drainage with the use of electrocoagulation may be resorted to.

HILL.

Medical Progress: Endoscopy.

Benedict, E. B.: New England J. Med., vol. 235, Nos. 1 and 2 (July 4 and 11) 1946.

This is a thorough summation of leading articles in the field of endoscopy including gastroscopy and peritoneoscopy. One hundred thirteen references are given.

HILL.

Physiology and Functional Pathology of the Lymphatic System Applied to Allergy of the Nose and Paranasal Sinuses.

Robison, J. Mathews: Journal of Allergy, March, 1946.

The author explains how the lymphatic system has been developed in the phylogenetic scale of life as it became necessary to remove certain body fluids and substances that are not readily absorbed by the bloodstream. Disorders that may result in pathologic changes can be the result of a failure of this lymphatic system to carry away stagnant extravascular fluid.

Since Dr. Robison's paper is on allergy of the nose and paranasal sinuses, he especially describes the characteristics of the lymphatic bed in the sinuses, directing particular attention to that of the maxillary sinuses and the ethmoids. An acute lymphatic blockage may result from a deposit of fibrin, cells, etc., in the interstitial lymph spaces, channels, and infected or malignant lymph nodes. Chronic lymph blockage results from fibrosis in the interstitial tissue spaces, lymph pathways, and nodes because of the prolonged presence of stagnant tissue fluid and bacterial products, parasites, or long-continued absorption of particulate matter. The stagnant tissue fluid renders the part very susceptible to recurrent infection.

The ability of the stroma of this mucosa to become edematous and then return to normal is a criterion of its physiologic efficiency and its complete recovery from infection or irritation. In acute bacterial sinusitis, the edema is great enough to set up a lymph pulsation through the dilated arteriovenous anastomoses. In chronic bacterial sinusitis, the proteinized fluid of the acute stage remains in

the dilated tissue spaces of the mucosa. This finally leads to a proliferation of the fibroblasts of the stroma and there is a permanent thickening and overgrowth of the part. "The result is a mucosa which can no longer become edematous and carry out its physiologic function of approaching the capacity of the sinus and returning to normal." This means that the sinus is unable to empty itself of its contained secretion. This type of sinus disease can be relieved markedly by irrigation, but there is a tendency to recurrence.

The author divides allergic maxillary sinusitis into three stages—acute, subacute, and chronic, and he states that the absorption of globulin, which is present in the membranes in a relatively high concentration, is likely to be at least one cause of an accompanying bronchial asthma.

McLaurin.

Notices

FIRST PAN-AMERICAN CONGRESS OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY OCTOBER 17-19, 1946

Immediately following and overlapping with the annual meeting of the Academy in October of this year, the First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology will take place, under the sponsorship of the Academy. This Congress will open with a banquet and evening session on Thursday night, October 17, and will continue on the two subsequent days. The first scientific session will be a joint session with the Otolaryngology Section of the Academy, and three additional sessions will take place on Friday afternoon, October 18, and Saturday morning and Saturday afternoon, October 19.

All interested members of the Academy are strongly urged to plan on remaining for the Pan-American Congress for which an excellent program is being arranged. The registration fee will be $5.00.

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Chicago at the Palmer House from October 8 to October 12. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5, Md.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Chicago, Ill., October 13-18, 1946.

American Broncho-Esophagological Association

President: Dr. Robert M. Lukens, 1001 Medical Arts Bldg., Philadelphia 2, Pa.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.

American Laryngological Association

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harry W. Lyman, Carleton Bldg., St. Louis 1, Mo.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

Sections:

Eastern—Chairman: Dr. Robert F. Ridpath, 1737 Chestnut St., Philadelphia 3, Pa.
Southern—Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Rd., Miami Beach, Fla.
Middle—Chairman: Dr. Oliver E. Van Alyea, 135 S. LaSalle St., Chicago 3, Ill.
Western—Chairman: Dr. Colby Hall, 1136 West 6th St., Los Angeles, Calif.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.

American Otological Society

President: Dr. William E. Grove, 324 E. Wisconsin Ave., Milwaukee 2, Wis.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.

First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, 3, Pa.
Meeting: Palmer House, Chicago, Ill., October 17-19, 1946.